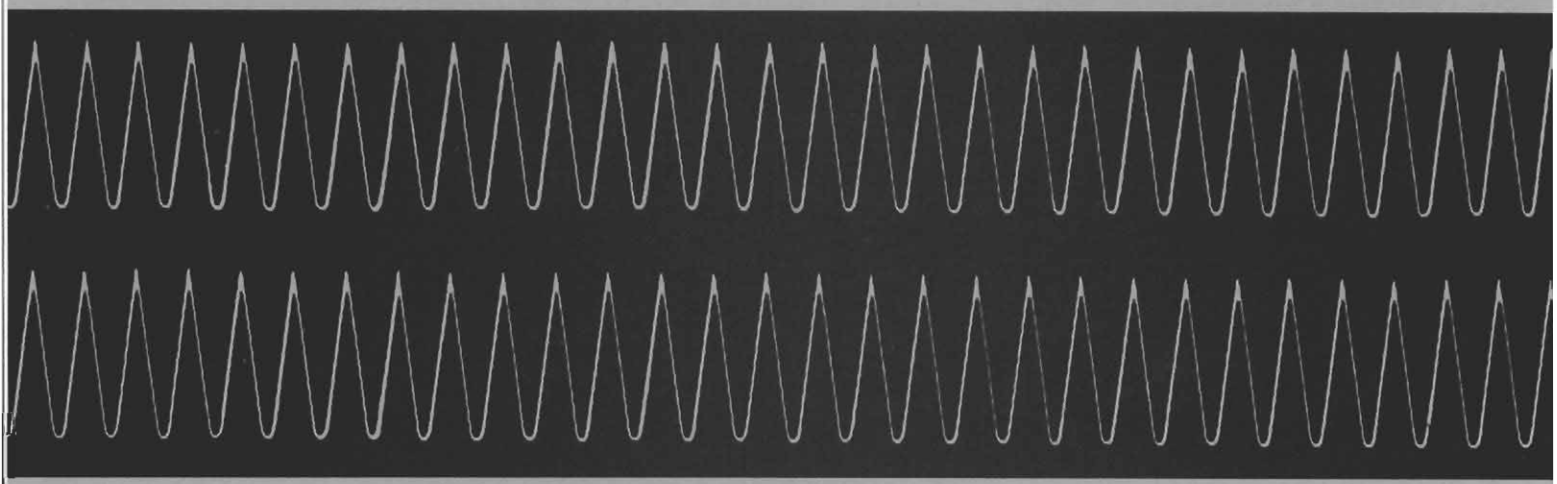
Research Needs in Stuttering: Roadblocks and Future Directions



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Edited by
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PREFACE

Many basic questions need to be answered concerning stuttering, encompassing etiology, development, characterization, and treatment. The definition of the disorder, means of identification, the determination of etiology and efficacious treatments, need to be resolved. To enhance knowledge about stuttering, we need to identify what factors would contribute to the advancement of stuttering research. To enhance federal support for stuttering research, we need to increase both the quality and number of grant applications submitted to the NIH on stuttering.

These needs led the Division of Communication Sciences and Disorders of the National Institute on Deafness and Other Communication Disorders, NIH, in September, 1988, to invite 12 clinicians and researchers to a two-day workshop. Prior to the workshop, participants were assigned specific topics and asked to develop position papers on current status, research needs, and future directions, in one of the areas of stuttering research listed below:

- Definition/subject selection criteria
- Measurement of stuttering severity
- Etiology
- Subtyping stutterers
- Pathophysiology
- Treatment

These position papers were discussed by the group, revised, and assembled for dissemination to the research community. Each chapter included in this volume represents not only the interests and opinions of the author, but also the incorporation of suggestions from the other participants during the workshop. A final chapter presents a summary of the discussions during the workshop, and reflects the comments, controversies, and questions raised on specific topics.

Although consensus was initially a goal and was in fact reached in some areas, it proved elusive in others, as the final chapter will reflect.

The issues raised in this volume identify the next logical steps that need to be taken, that can be taken, in stuttering research. It remains for the research community to accept the challenge. It is hoped that this document will engender increased interest in stuttering and assist investigators in developing studies to increase our understanding and treatment of this disorder.

Judith A. Cooper, Ph.D

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A. DEFINITION AND IDENTIFICATION OF STUTTERING

Chapter 1

CHILDHOOD STUTTERING: WHAT IS IT AND WHO DOES IT?

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The purpose of this chapter is to discuss the current state of affairs with regard to the definition of childhood stuttering and the classification of young speakers as stutterers. While some of this discussion may be similar to that pertaining to adults, much of it is unique to children, for example, the overlap in the number and nature of speech disfluencies between normally fluent and stuttering children. Although we will note roadblocks to research with young stutterers due to problems with definition and categorization, we will also make suggestions for changes in methodology as well as future directions for research. As we will try to show, our ability to define stuttering and categorize stutterers impacts nearly every facet of our study of stuttering in childhood.

DEFINING STUTTERING

State of the Art

Speech, like many other behaviors, is occasionally produced, by all speakers with hesitations, interruptions, prolongations, and repetitions. These disruptions in the fluent or forward flow of ongoing speech behavior are termed *disfluency* and their frequency, duration, type, and severity vary greatly from person to person and from speaking situation to speaking situation. Some of these speech disfluencies, particularly those which involve within-word disruptions such as sound or syllable repetitions, are most apt to be classified or judged by listeners as *stuttering* (e.g., Boehmler, 1958; Schiavetti, 1975; Williams & Kent, 1958; Zebrowski & Conture, 1989).

Definition of terms. Given that listeners typically judge within-word speech disfluencies as stuttering, we will define, for the purpose of this chapter, *stuttering* or *stuttered* speech as any within-word speech disfluency, for example, sound/syllable repetitions, sound prolongations, broken words, and so forth (cf. Johnson, Darley, & Spriesterbach, 1963, pp. 209–210). (This does not deny the importance of disruptions in rate, pitch, loudness, facial gestures, and the like to a description of stuttering; however, for the purposes of this discussion, these disruptions will not be viewed as the sine qua non of stuttered *speech* and will instead be considered as events *associated* with stuttered speech.) *Fluency* or *fluent* speech will be defined, for the purposes of our discussion, as *speech whose rate, rhythm and forward flow is free from any overt hesitations, repetitions, prolongations, interruptions, or stoppages.* Some of these "hesitations, repetitions ..." (i.e., disfluencies) in fluent speech consist of within-word disfluencies or stutterings (e.g., sound/syllable repetitions) while others consist of such between-word disfluencies as revisions, phrase repetitions, interjections, and so forth (i.e., "normal" disfluencies). As we will see, there is considerable "overlap" in the number of between- as well as within-word disfluencies between children considered to be normally fluent and those considered to be stutterers, particularly during early childhood.

Terms typically used to describe various aspects of stuttering and disfluency—*frequency, duration, severity, disfluency type,* and *associated behavior*—will also be defined as they are used in this discussion. *Frequency* of stuttering refers to the number of instances of stuttering per some unit of speech, usually 100 words or syllables of reading or conversational speech. *Duration* of stuttering refers to the temporal length, in milliseconds or seconds, of an instance of stuttering, usually averaged over a randomly selected sample of several instances of stuttering within a reading or conversation. *Severity* of stuttering refers to the subjective, rather holistic, judgment of the degree of stuttering exhibited by a stutterer, usually expressed in terms of *mild, moderate,* or *severe* and relates to the stutterers' problem as a whole but can also be applied to separate instances of stuttering (cf. Sherman & McDermott, 1958). *Type* of speech disfluency refers to the various within- or between-word hesitations, interruptions, pauses, prolongations, repetitions, and stoppages that characterize 'stutterers and nonstutterers' speech (cf. Johnson, Darley, & Spriesterbach, 1963, pp. 209–210). *Associated behavior* refers to those speech and

nonspeech behaviors (cf. Schwartz & Conture, 1988) that occur relatively consistently during instances of stuttering or within-word disfluencies, for example, changes in pitch, blinking of the eyes, covering the mouth with the hand, and so forth.

In this section we will make two major points with regard to the definition of stuttering in children: (a) there are *no known objective, listener-independent* criteria for identifying instances of stuttering or classifying children as stutterers versus normally fluent speakers and (b) there is *no consensus* among experienced clinicians and researchers regarding behavioral definitions of stuttering in childhood or classification of children as stutterers.

No known objective, listener-independent criteria. Presently, there are no known objective, listener-independent criteria for distinguishing between instances of stuttering and instances of other types of disfluency or for classifying which young talkers are stutterers. As Young (1984) notes, there is "... no test within science which can determine once and for all whether a fluency departure is a stuttering instance or a nonstuttering disfluency" (p. 13). Bloodstein (1987) states that, "... the identification of moments of stuttering always involves the judgment of a listener" (p. 4). Similarly, Young (1984) says that the "... ultimate detection and measurement instrument for stuttering and stutterers is a human observer, as it should be, since 'stuttering' and 'stutterers' represent human judgments" (p. 28). Young (1984) further states that "All tools of measurement, both acoustical and physiological, eventually must be validated against the judgments of human observers" (p. 28). Ironically, even though listener judgments and labelling of certain disfluencies as stuttering are subjective, these judgments and labels are relatively consistent within and between trained judges. Agreement among observers is generally higher, however, for total instances of stuttering than for unit-to-unit or identical instances of stuttering (e.g., Curlee, 1981).

As Conture and Schwartz (1984) note, "... it is still unclear whether such labeling, by listeners, reflects (a) listeners' unconscious intuitions regarding speakers' speech behaviors that do and do not indicate a speech problem or (b) listeners' learned, perhaps culturally determined, intolerance for certain forms of speakers' speech behavior" (p. 1). Despite theoretical concerns about the reliability and validity of these judgments, listeners have and will continue to make such judgments until the professional community begins to provide some guidance in the form of definitions of childhood stutterings and stutterers based on consensus.

Lack of consensus regarding behavioral definitions. Related to the fact that our definitions of stuttering in childhood are subjective as well as listener-dependent is the fact that experienced clinicians and researchers alike have not reached a consensus on behavioral definitions of childhood stuttering. It is this writer's opinion that professionals who are involved with stutterers, from either a clinical or research point of view, now have enough information to begin developing a consensus definition of instances of stuttering in children. Arriving at consensus will not solve all problems of definition and terminology but it should help these same professionals more clearly, precisely and reliably communicate between themselves regarding childhood stuttering. It should also help these workers develop less ambiguous measurement and subject selection procedures as well as clearer and more precise reporting of same in scholarly journals and professional conferences.

Needed Knowledge and Research

The above description of the state of the art regarding the definition of stuttering in childhood is suggestive of areas where further knowledge and research is needed. It is not our purpose in the following section to specifically design studies as much as to suggest areas where knowledge is needed and/or where further research might be productive. While some of these studies would be experimental others would be descriptive but all should advance, to greater or lesser degrees, our understanding of how to describe and define instances of stuttering in young speakers.

Definitions based on clear, precise and intention-free terminology. Terminology is needed to more clearly and precisely describe instances of stuttering as well as other speech disfluencies. This is not a new concern; Wingate (1964) voiced the same concern when postulating guidelines for the definition of stuttering. Old or new, however, the problem of unclear, imprecise terminology for describing stuttering and stutterers still remains. At least two criteria must be met when developing terminology that clearly and precisely defines and describes stuttered speech. First, terms used to define, talk and write about instances of stuttering should be, as much as possible, stated on a descriptive level of verbal abstraction (cf. Johnson, 1946, pp. 127–142). This involves descriptions based on direct observations (i.e., first-order verbal abstractions) of speech and related behavior, for example, "He exhibited 3 sound/syllable repetitions per 100 words of conversational speech." Such descriptions are preferable to abstracting of inferences based on descriptions (i.e., a second- or third-order verbal abstraction), for example, "He is repeating because he is nervous." Second, such terminology should be intention-free, that is, eschew interpretation in favor of description of behavior. It will be very difficult to achieve clear and precise terminology as long as we mix our descriptions based on direct observations of behavior together with our idiosyncratic interpretations of the individual's supposed reasons or intention for exhibiting the behavior.

At present, many terms are used which have been borrowed from clinical practice where their use is more utilitarian for communication with lay clients than for the purpose of research. Descriptive terms, like "sound/syllable repetition," are based on observation. They indicate that a sound or syllable is reiterated and do so without allusions to the speaker's reasons or intention for producing it. Other terms, however, like "starter," "filler," or "block," are not only imprecise descriptors of what

the speech behavior sounded or looked like but they also allude to or make interpretations of the stutterer's supposed reasons for the production of the utterance. Furthermore, terms like *block* or *tense pause* are expressed at more inferential levels of verbal abstraction than descriptive terms such as *sound/syllable repetition*. While more inferential or abstract terms (e.g., *fillers* or *starters*) may have usefulness in certain clinical settings, the purposes of empirical research require clear, precise and intention-free descriptions of behavior (e.g., sound/syllable repetitions).

Objective correlates of subjective listener judgments. Realizing that listeners are the only real judges of whether a particular instance of disfluency is stuttered should not deter us from assessing which, if any, objective measures may correlate with listener judgments of childhood stuttering. While some might argue that it doesn't matter whether we understand the basis for judges' perceptions of young stutterers' speech as long as the judges are internally and externally in agreement, it does matter when trying to devise tests of stuttering severity that have clinical as well as experimental usefulness, when instructing judges what they should be basing their judgments upon or when training student-clinicians or clinical scientists.

Such objective measures could take a variety of forms: acoustic measures of duration, intensity or frequency components of speech; level of physical tension in speech musculature; onsets and offsets of movements of oral structures; or pressure/flow measures associated with speech production. When describing young stutterers' stutterings, experimenters should, at the very least, routinely specify both the type of measured speech disfluency and associated phonetic features. Such specification would go a long way towards developing a useful, objective index of stuttering for the purposes of descriptive as well as experimental research.

One interesting attempt to make objective measures of stuttering and correlate them with listener judgments was reported by Howell, Hamilton, & Kryiacopoulos (1986). Acoustic representations of stutterers' repetitions and prolongations were "automatically recognized" by means of computer algorithms. Howell et al. (1986) considered not only the characteristics of the various instances of stuttering but also the associated phonetic elements and reported 100% computer recognition of 8 repetitions and 70% recognition of 7 prolongations. However, as other acoustic studies of stutterers' stutterings point out (e.g., Howell & Vause, 1984; Howell, Williams, & Vause, 1987; Kelly & Conture, 1988), the acoustic characteristics of stuttered speech make it highly unlikely that *all* instances and types of stuttering will be accounted for by a *single* algorithm or objective measure.

Until we know which, if any, objective measures are reliably associated with listener judgments of instances of stuttering in children, definitions of stuttering in childhood must rely on the judgments of trained observers who agree with the judgments of other trained observers. This is problematic because it continues to place listeners in the position of judging whether something has or has not occurred. Particularly in those studies where changes in stuttering behavior are the basic data, listener bias can be a tremendous factor. Indeed, pre- versus post-therapy studies are difficult enough due to such variables as the Hawthorne effect (cf. Homans, 1965; that is, people changing their behavior simply because they know they are in a study or because experimenters pay attention to them) without have to contend with uncontrolled listener bias as well. Ideally, such objective measures would have research applications as well as clinical utility. That is, the methods needed to make such measures would be based on events and behavior a clinician can directly and readily observe, collect, and interpret.

Average and range of disfluencies in normally fluent youngsters. Research is needed to specify the number and variety of speech disfluencies that occur during typical conversations of children between 2 and 7 years of age. While Johnson (1959) and his colleagues' data are of tremendous assistance in this regard (and, in recent years, those of Yairi 1981, 1982; Yairi & Lewis, 1984), they still do not make clear what the central tendencies and variability of speech disfluencies are for 2-year-olds, 3-year-olds, and so forth. Without this information it is hard to assess the extent to which a child suspected or known to be a stutterer deviates from his or her age norms or how closely an individual normally fluent child approximates them. Although our information on the characteristics of young stutterers is improving, for example, the *Stuttering Severity Instrument* (Riley, 1980), we are still less than clear how these characteristics compare with the population of normally fluent children.

Relation of changes in time/tension of speech production to listeners' judgments of speech disfluency. Research is also needed to assess the influence of time on listeners' perceptions of young stutterers' stutterings. Because it has been speculated that stutterings are related, at least in part, to disruptions of the physical tension/temporal aspects of speech production (e.g., Conture, Colton, & Gleason, 1988; Starkweather, 1987, p. 143–154; Van Riper, 1971, 1982), it seems important to understand how changes in one or both of these variables—time or physical tension—are most clearly related to listener judgments of stuttering. Listener perceptions of stuttering may be as much related to their sense that "too much time has been taken up" by an instance of stuttering as it is to their perception that the sound, syllable, or word was reiterated or prolonged "with too much tension."

Indeed, Franken's (1987, 1988) perceptual rating scales used to judge various aspects of stutterers' speech (e.g., naturalness, speaking rate, voice quality, etc.), suggest that "tempo" is highly related to listeners' perception of differences in stutterers' pre- versus post-therapy speech. Franken's observations are consistent with Prosek & Runyan's (1983) finding that manipulations of phonetic-segment and pause duration influenced listeners' discrimination of treated stutterers from nonstutterers. Prosek and Runyan (1982) had previously reported that speaking rate and pauses also influence listeners' ability to differentiate stutterers' fluent speech (i.e., speech con-

taining no overt stutterings) from that of nonstutterers. In summary, changes in the temporal aspects of speech production appear to be associated with listeners' perceptions of treated adult stutterers' and this type of research is also needed to assess the speech of treated children who stutter.

Consensus definitions of stuttering in children. A consensus definition of stuttering in childhood is needed. A consensus definition is only possible, however, if we realize that identification of instances of stuttering is based on human judgment and is not the result of some physical or natural law. Considering certain speech disfluencies as "stuttered" and others as "normal" would be a bit like the arbitrary borders used to mark the beginning of one state and the end of another. For example, on one side is Vermont and on the other is New York, not because of natural law or differences but because custom, convention, and arbitrary but agreed-upon law so decree.

A consensus definition would help experimenters and clinicians come to an agreement regarding those speech disfluencies produced by children that they consider as "stuttered" and those that they call "normal," regardless of the basic arbitrariness of this agreement. If such agreement took place, this would tell others, at the very least, what we are talking about when we label as "stuttered" selected aspects of a child's speech behavior. Agreement would lead to better communication, less argument over whether "stuttering" did or did not occur and a greater chance for independent investigators to replicate findings.

Liabilities of consensus definition of stuttering in children. It is unclear whether behavioral definitions of stuttering would increase or decrease interobserver agreement on either total frequency of stuttering or on specific words stuttered (unit-by-unit) based on the somewhat contradictory findings of Young (1975) and Martin and Haroldson (1981). Young (1975) found more instances of stuttering marked under the "stuttering-undefined" condition whereas Martin and Haroldson (1981) found less stuttering marked under that condition. Thus, findings suggest that increasing agreement on stuttering between observers is not as simple as merely giving all observers the same behavioral definition of stuttering.

Even with a consensus definition of stuttering in childhood, some instances of stuttering are probably going to be difficult to readily and reliably perceive because of their brief, inaudible, and nonvisible nature, for example, short, inaudible sound prolongations. Furthermore, certain disfluency types may more closely mimic or resemble nonstuttered types of speech disfluency than other disfluency types. For instance, sound/syllable repetitions may be easier for judges to agree on an instance of stuttering than sound prolongations (cf. Zebrowski & Conture, 1989).

DEFINING WHO IS A STUTTERER

State of the Art

On the surface, it seems that categorizing a child as a stutterer is simple. However, because the definition of stuttering itself is still unclear, our definition of stutterer must also remain unclear. As Young (1984) points out, however, even "Untrained observers (can) make a clear distinction between stuttering and stutterer, using the former classification more frequently than the latter label, and believe that an individual can stutter without also being a stutterer" (p. 27). Thus, instances of stuttering are necessary but not sufficient for an individual to be classified as a stutterer.

The biggest problem with differentiating children who stutter from those who don't is the fact that there is an overlap in the number and nature of speech disfluencies exhibited by the two talker groups. However, if we study the speech disfluencies of one of the largest available samples of young stutterers (N = 89) and their normally fluent peers (N = 89) (Johnson et al., 1959), we see that even though there is overlap, the number and nature of speech disfluencies produced by the two groups are not carbon copies of one another. In fact, data from the Johnson et al. (1959) study shows that young stutterers (a) produce more speech disfluencies than their normally fluent peers and (b) are much more apt than their normally fluent peers to produce certain types of speech disfluency. For example, Johnson et al. (1959) reported that 70% of children labeled as normally fluent produce 1.0 or less *within- word* speech disfluencies, while only 20% of children labeled as stutterers produce so few. Johnson et al. (1959) also reported that 50–60% of stuttering children produce 3.0 or more within-word disfluencies, while less than 10% of normally fluent youngsters do so. Similarly, Yairi and Lewis (1984) reported that part-word repetitions were the most frequent type of speech disfluency produced by 10 2- to 3-year-old stutterers within 2 months of the onset of their problem (part-word repetitions being very infrequently produced by normally fluent children).

In essence, there is far less overlap between young stutterers and normally fluent talkers in the frequency of their *within- word* disfluencies. Even so, there is no known behavior, speech or otherwise, that young stutterers exhibit that young nonstutterers *never* exhibit. There is no published evidence that the speech disfluency of young stutterers' disfluency is *categorically* different from that of their normally fluent peers.

Deciding who is and who is not a stutterer must necessarily, therefore, be based on *relative* versus *absolute* criteria. Ideally, as was discussed with the definition of stuttering, these criteria would be (a) intention-free, amenable to external observation, and objectively measurable and (b) descriptive of speech and associated nonspeech behavior. Whatever criteria are used, research is needed to determine the relative frequency and types of speech disfluencies which a child can exhibit and still have a *high probability* of being considered a normally fluent speaker. Based on what we have discussed, it would appear unrealistic to expect to develop criteria which could be used, with total certainty, to judge who is and who is not a stutterer. What we are talking about here are statements of probability rather than statements of

certainty. It is not the mere presence but the relative amount of frequency and type of a child's speech disfluency that help us to decide whether the child should be considered a "stutterer." What is missing, however, are some guidelines for determining when the probability is high that a particular frequency or type of disfluency is or is not of concern (i.e., is or is not sufficient grounds to say that a child is *at risk* for developing a stuttering problem).

A variety of tests are used to assess the speech and related behaviors of children known or suspected to be stutterers (Brutten, 1982; Guitar & Peters, 1980; Johnson, Darley, & Spriesterbach, 1963; Riley, 1980; Riley, 1981; Stocker, 1976; see Conture & Caruso, 1978 for review of Stocker, 1976; Thompson 1983), but most of these attempt to qualitatively and/or quantitatively specify the *degree* or *severity* of the stutterer's problem rather than differentiate stutterers from nonstutterers. In essence, most of these tests assume that the individual under consideration *is* a stutterer (with the only question being the "degree," "severity," or "type" of stuttering). However, at least two of these tests (the Iowa Scale and the Stuttering Severity Instrument) do provide some information that permits comparison to speech behavior expected from the normally fluent speaker (cf. Conture & Caruso, 1987, pp. 89–90 for more detailed discussion of these tests). Furthermore, attempts have been made to refine scaling procedures for assessing, for example, the severity of stuttering (cf. Schiavetti, Sacco, Metz, & Sitler, 1983), but, as noted above, the criteria that underlie judges' decisions about stuttering are still less than clear.

In summary, a listener is most apt to judge a child to be a stutterer if that youngster exhibits enough of the types of behaviors that the listener judges to be stuttering! In essence, there is no purely objective means for determining whether a child is a stutterer any more than there is for deciding which sound, syllable, or word is stuttered. Listeners can be and are trained, however, to make this judgment with a high degree of internal agreement as well as agreement with others.

Needed Knowledge and Research

The above description of the state of the art regarding categorizing children as stutterers is suggestive of areas where further knowledge and research is needed. It is not our purpose in the following section to specifically design studies as much as to suggest areas where knowledge is needed and/or where further research might be productive. Although some of these studies would be experimental others would be descriptive but all should advance, to greater or lesser degrees, our understanding of how to best differentiate children who stutter from those who don't.

Determining the basis on which experts make their subjective judgments. It would be very helpful to know the basis on which experts in the area of stuttering are able to arrive at their judgments of which children are and are not stutterers. Obviously, these experts are basing their decision on a variety of acoustically as well as visually apparent aspects of speech and related nonspeech behaviors. Thus, experts may use a complex combination of visual and auditory information regarding a child's speech together with linguistic, attitudinal, cognitive, and emotional features exhibited by the child to arrive at their judgments of who is and who is not a stutterer. Perhaps, however, within this complex of information, only one or two objective measures of speech behavior are highly and consistently correlated with experts' judgments. Knowing more about the existence and nature of these measures would seemingly help experimenters and clinicians develop a more reliable, objective, and streamlined means for determining who is and who is not a stutterer and one that would be highly correlated with the judgments of experts.

Variations in young stutterers's stutterings. It would also be helpful to know how subjective and objective assessments of stutterers' stutterings vary over time. For example if one were to sample, across days or weeks, a particular stutterer's stuttering what sorts of variation might one expect between the various samples? Very little is known about variations in the type, duration, frequency, and severity of young stutterers' stutterings across time. Parents tell us that their youngsters' stutterings vary and it seems reasonable to speculate that the greater the absolute frequency of stuttering, the greater the magnitude of absolute variation would appear to casual observers. However, we do not know whether these variations are periodic, quasi-periodic, or aperiodic. Based on our clinical experience, we suspect that variations in young stutterers' are essentially aperiodic because the factors that influence this variation are so great in number and highly interactive.

It would be very instructive to have researchers collect data on, say 30 or so young stutterers at or near the onset of their problem and then follow them for 5 to 10 years during which time they receive *no* speech and language therapy. However, ethical and legal restrictions on withholding services would appear to preclude the possibility of such a study. This is particularly of concern with young children because various clinicians (e.g., Conture, 1990; Gregory & Hill, 1984; Starkweather, 1987) believe early, (i.e., nondelayed) intervention is important for maximal benefits.

Whatever the case, knowing more about variations in youngsters' speech disfluencies would help clinicians and clinical scientists better compare changes associated with therapy with other changes that typically occur over a similar timeframe. Having more objective information regarding expected variations in stuttering would be particularly helpful when trying to assess a child whose frequency of stuttering is at or near the "cut-off" for being considered a normally fluent speaker. These are the children who on Monday may exhibit a frequency of speech disfluency that is within and on Tuesday outside of normal limits.

Consensus definition of stutterer. Researchers and clinicians could reach consensus regarding a classification scheme for deciding which children are and are not stutterers. There is sufficient data (e.g., Johnson et al.,

1959) available to show that certain frequencies of selected disfluency types are more apt to be produced by young stutterers than their normally fluent peers; however, we again note that consensus definitions would have to be based on convention and arbitrary categories. Further, there would always be the false positives (i.e., children whose disfluencies are actually at the high end of the normal range but who are considered to be stutterers) and false negatives (i.e., children who are stuttering but considered to be normally fluent (cf. Conture, 1990, p. 11–12). Even more than with definitions of stuttering, classification of stutterer versus nonstutterer appears to require binary or categorical labels to describe what appears to be a fluid or continuous distribution of behaviors *and* "behavers." It is a little bit like trying to paint stripes on a gravel-covered parking lot in attempts to mark off parking spaces. Although our parking spaces might, at least initially, contain equal space and remain equally distributed throughout the lot, they would soon begin to unpredictably change shape and size because the "categorizing" stripes were applied to a continuously changing and shifting surface.

Reaching a consensus to call a child a "stutterer" requires us, at least for the foreseeable future, to rely on human judgment. However, whether or not a consensus is reached, lay people, clinicians, and researchers alike will continue to judge and label certain children as stutterers even though they lack a modicum of guidelines for doing so. Is it not better to arrive at some reasonable consensus for making these judgments than to capitulate to the belief that consensus is impossible because we seldom obtain complete agreement between and within human judgments?

Consensus agreement need not be absolute. Means and ranges of criteria measures can be stated and children can be considered not just as a "stutterer" or a "nonstutterer" but inside or outside of normal limits or at the lower or upper ends of normal limits or at no, low, medium, or high risk for stuttering. Once again, whether consensus is reached, such decisions are made daily by many clinicians and researchers with few widely accepted guidelines. Stuttering, however, is not a local phenomenon but one that spans the USA and the world and, with a few exceptions (e.g., Bloodstein's 1987, p. 253, discussion of Afrikaans-speaking stutterers' tendency to stutter on word-initial vowels which are typically produced, in the Afrikaans language, with a hard attack), its characteristics are fairly universal. Thus, definitions of who is a stutterer and what is stuttering should not be a local option but an agreed-upon convention that could reasonably apply across a wide variety of settings and for the greatest numbers of stutterers possible.

Liabilities of a consensus means of categorizing children who stutter. The first liability of having consensus on how to categorize children as stutterers is that this may be a unidimensional answer to a multidimensional problem. Stutterers exhibit more than just disruptions in speech prior to and during instances of stuttering. This complex of speech and nonspeech behavior might be inappropriately described or overlooked if the classification were too restricted or unidimensional in nature.

The second liability of having a consensus means of categorizing young stutterers is that certain groups of young stutterers, for example, children who only produce, but consistently so, 1 or 2 within-word disfluencies per 100 words, might not fall within the observable behavioral criteria for classifying who is or who is not a stutterer. At this point, it is unknown whether these "sub-clinical" or potential young stutterers gradually or quickly become normally fluent or whether they become full-fledged stutterers.

A third liability is that consensus definitions, particularly those that are overly rigid, might exclude some youngsters who often wander back and forth across an arbitrarily-agreed-upon border between normal fluency and stuttering. This liability is potentially quite serious because we know that the stuttering of young children waxes and wanes in a relatively unpredictable fashion. We therefore would not want our definition of who is and who is not a stutterer to be so rigid that it would classify a child as a stutterer who is more often normally fluent than stuttering or, conversely, to classify a child as normally fluent who is more often stuttering.

A fourth liability is that any relatively rigid, unifying definition for classifying speakers as stutterers might overlook important behavioral differences *between* young stutterers that may have a great deal of significance for clinical as well as research purposes (cf. Preus, 1981; Schwartz & Conture, 1988). Future research may show that differences *between* young stutterers are just as varied and numerous as, and perhaps more important than, differences between young stutterers and their normally fluent peers.

IDENTIFICATION MEASURES

What Childhood Speech Behavior Should be Considered "Stuttered"?

Clinicians typically use the following aspects of disfluent and related speech behavior when trying assessing stuttering in children: (a) overall frequency of all speech disfluencies (between-word plus within-word); (b) percent of all spoken words and/or percent of all speech disfluencies which are within-word; (c) average duration of instances of stuttering: (d) informal as well as more formal assessment of stuttering severity; and (e) nature and number of associated speech and nonspeech behaviors. Various guidelines for the use of these behaviors when assessing stuttering in children known or suspected to be stutterers have been discussed elsewhere (e.g., Adams, 1980; Ainsworth & Fraser, 1988; Gregory & Hill, 1984) and this discussion will not be duplicated in this space. Instead, we will discuss principles that we think should underlie the measurement of stuttering and related behavior in children. Briefly, these measurements should be: (a) *reliable* between and within independent observers, (b) based on subject-independent or *external observations*, (c)

sufficiently *variable* to permit differentiation between and within stutterers, and (d) emphasize *objective* measures rather than *subjective* impressions.

First, identification methods should be *reliable* and replicable between and within judges. Although a particular clinician or researcher may be quite internally consistent when identifying which young children are stutterers, it should be possible for appropriately experienced and trained independent judges to make reasonably similar identifications when observing comparable subjects. Ideally, the Sander's (1961) agreement index between- and within-judges should be 0.80 or greater *and* the between- or within-judge measurement error appreciably smaller than any reported experimental effect or between- or within-group differences. At the least, clinicians and researchers should be able to communicate the basis for his or her judgments so that their special knowledge can be passed on to other professionals or the next generation of researchers and clinicians.

Second, the measure under consideration must be sufficiently *variable* to permit differentiation between subjects as well as detection of changes in subjects associated with experimental and therapeutic procedures (we will return to this issue below). If a behavior exhibits little variation from one stutterer to the next, regardless of circumstances, than it is probably not a very useful behavioral measure.

Third, the measure must be *externally observable* to people other than the stutterer. Although stutterers' feelings of anticipation or expectancy to stutter are certainly a reality to the stutterer, the presence or absence of these feelings and attitudes are not easily identified by external listeners. Furthermore, these feelings and attitudes seem to have little relation to at least some objective measures of speech (cf. Kelly & Conture, 1988). This does not imply that clinicians should deny or disregard their young clients' or their parents' descriptions of behaviors or feelings but neither should such descriptions serve the *sole* basis for classifying a youngster as a stutterer. As independent, problem-solving clinicians and researchers, we should strive to be able to produce accurate, reliable, and replicable records of our young stuttering clients' behavior rather than *solely* basing our definitions of "stutterer" on the young subjects' and/or their parents' verbally expressed reports.

Fourth, and finally, the measure should be *objective* to the point that a number or set of numbers can be assigned to it—whether this is a percentage, number per sample, or scale value. Thus, the ideal measure of stuttering would be replicable and reliable within and between judges, be sufficiently variable to permit differentiation among stutterers, be externally observable and objective enough to permit numbers of an ordinal or beyond level of measurement.

What Subject Characteristics Should be Considered When Deciding Which Young Talkers are Stutterers?

When studying children who stutter, researchers may want to control for cognitive, psychological, or physical variables such as intelligence, social adjustment, neuromotor abilities and the like to minimize their influence on their findings. It should be realized, however, that there is presently nothing to suggest which, if any, of these other variables should be considered when trying to identify who is and who is not a stutterer. Stutterers' apparent similarity to normally fluent speakers on these other variables strongly suggests that these variables are of minimal use when classifying individuals who stutter and that speech-related variables are still the most useful in this regard (cf. Schwartz & Conture, 1988). One exception to this would appear to be the child's chronological as well as developmental age in relation to exhibited speech behaviors, for example, the frequency of sound/syllable repetitions. What is "acceptable" fluency and "unacceptable" disfluency probably differ for a 4-year-old as opposed to a 7-year-old but although this seems apparent we still know very little about the relation of chronological and/or developmental age to children's speech disfluencies. In essence, the same criteria used to define stuttering (i.e., Is the measure appropriately reliable and replicable, variable, externally observable and objective?) should be applied when trying to classify children as stutterers or normally fluent speakers.

In an ideal world, our criteria for identifying who is or who is not a stutterer should be highly related to a young stutterers' own internal feelings that they are a stutterer. However, children are less apt than adults to verbally explicate their internal feelings about their speech. At present, therefore, clinicians and researchers must rely on external measures of stuttering rather than young stutterers' verbal reports about themselves and their speech. Indeed, more will probably be learned about the nature, number, and variability of young stutterers' internal feelings about stuttering when we can compare them to definitions of stuttering and stutterers that a majority of external observers have agreed upon.

DIFFERENTIATING "STUTTERED" FROM "NORMAL" DISFLUENCY

Clinical Applications

In our clinical experience, one reasonable initial screening device is the total frequency of speech disfluency. Adams (1980), for example, suggests that an overall frequency of 10% or more, regardless of disfluency type, is useful in distinguishing between children at risk for stuttering and those more likely to be normally fluent. We concur that 10% overall disfluency is a useful benchmark to decide whether a child is or is not stuttering; however, we also try to decide whether the child produces 3 or more *within-word* disfluencies per 100 words of conversational speech. That is, any child who exhibits both 10% overall disfluency *and* who produces 3 or more within-

word disfluencies per 100 words is, based on our clinical experience, a child who is highly likely to be at risk for stuttering.

Once a child appears clearly at risk for stuttering, than other variables such as nonspeech associated behavior become relevant. These associated behaviors may help clinicians differentiate between young stutterers who should receive immediate therapeutic intervention and those who should be monitored by means of follow-up re-evaluations. For example, young stutterers can be distinguished into five statistically significant subgroups on the basis of the number of different behaviors as well as nonspeech behaviors associated with 10 of their stutterings (Schwartz & Conture, 1988). Knowledge of these five subgroups of young stutterers, based on associated behavior, may eventually find application to the differential diagnosis of stuttering in children. Again it should be pointed out that speech disfluency and associated nonspeech events are behaviors that change over time. Thus, a particular young stutterer may produce 10 stutterings per 100 words today but tomorrow produce anywhere between 3 and 23 stutterings per 100 words. Thus, the clinician, just like the researcher, should obtain not only the child's *average* amount or frequency of stuttering but some *index of its dispersion,* for example, the range.

Clinicians do not find it difficult to decide that a child is "normally fluent" if he or she exhibits extremely fluent speech. (In this writer's experience, this is speech containing 1.0 or less within-word disfluencies per 100 words). Likewise, it is not hard for the clinician to decide that a child is a "stutterer" if he or she produces 10 or more stutterings per 100 words spoken. It is, however, hard for clinicians to decide about a child whose behavior falls between those youngsters who can obviously be classified as normally fluent and those youngsters who can obviously be classified as stutterers. Unfortunately, these "in-between" youngsters represent a sizable portion of all children who stutter. In our clinical experience, these in-between children seem to come in three forms: (a) low or no risk of stuttering, (b) some risk of stuttering, and (c) moderate risk of stuttering. Tests like the Stuttering Severity Instrument (Riley, 1980) or Stuttering Prediction Instrument (Riley, 1981) help but are still less than adequate, in this writer's experience, when describing these in-between children. This is particularly true when trying to classify a child as a "stutterer" or "normally fluent speaker" when the child is producing (a) a small but consistent number of sound/syllable repetitions per 100 words in a physically effortless and/or relatively relaxed fashion, (b) few apparent associated nonspeech behaviors and (c) little or no verbal or nonverbal indications that he or she has emotional/intellectual awareness of his or her speech disfluencies.

Research Applications

Any researcher interested in studying stuttering in children *must* be concerned with whether he or she is studying (a) within- versus between-word speech disfluency and/or (b) children who stutter versus those who are normally fluent. Although most researchers now recognize that listeners typically judge within-word disfluencies as "stuttered," it is not quite as easy for researchers to distinguish between children who are normally fluent and those who stutter. That is, there is always the chance that children that researchers consider as "stutterers" may, with time, become normally fluent and vice versa.

At present, although figures vary, it would seem that somewhere between 50% to 80% of those children originally diagnosed as stuttering become normally fluent (Ingham, 1985; Sheehan & Martyn, 1970). Thus, researchers cannot be *absolutely* certain that those children they consider to be stutterers are and/or will remain so. Furthermore, much of our present information, for example, Sheehan and Martyn (1970), regarding recovery from stuttering is based on retrospective verbal or written reports of teenagers or young adults. Although such data cannot be dismissed out of hand, verbal or written recollections of past events and behavior would not seem to have the same degree of face validity as information gathered from direct observation of young stutterers over a period of years. Researchers who base their understanding of recovery from stuttering during childhood on the verbal reports of young adults' recollections of their past, should remember that these young adults must rely on their memories for these recollections. It is this author's clinical experience, however, that parents' and adult stutterers' recollection about the past, particularly the time, place, and events surrounding the onset of stuttering, is often clouded by the passage of time (cf. Conture, 1982, p. 158–163).

VARIABILITY

Variations in Type, Frequency and Severity of Stuttering

The frequency of stuttering in children varies in a relatively unpredictable fashion (Ainsworth & Fraser, 1988, p. 22; Conture, 1987, pp. 25–26). And, as Robinson (1964) has mentioned, it is the apparent random waxing and waning of speech disfluencies that is one of the more confusing and disheartening aspects of stuttering for the child and his or her parents. It is our observation, and one with which parents generally agree, that the child seems more disfluent when fatigued, answering or asking questions, excited or talking to inattentive listeners (cf. Davis, 1940). Because of such variability, central tendencies *must* be accompanied by indexes of dispersion in order to most closely circumscribe the child's speech behavior.

Although these variations make it difficult to clinically manage or empirically study childhood stuttering, changes in the frequency, type, duration, and severity of stuttering are part of the reality of the problem. Although there is little data to support our claim, it is our observation that the *frequency* of the child's overall disfluency and within-word disfluency is the most variable of all

measures of stuttering. In our clinical and research experience, variations in frequency by as much as 25 to 50% are not uncommon in children who stutter.

The young stutterer's *type(s)* of disfluency is somewhat less variable. That is, it is our experience that the child's most frequent disfluency type, for example, sound/syllable repetition, remains relatively the same until the child's stuttering problem worsens, at which point there is a gradual change in the most frequently exhibited disfluency type. Generally, in our experience, as the problem "worsens," the child changes from a *reiterative*-movement to a *stabilization*-of-movement type of speech disfluency (cf. Conture, 1990, pp. 23–26).

Duration of stuttering does vary between and within stutterers but its variation is not as readily apparent, at least perceptually, because its variation is in fractions of seconds or milliseconds. Changes in the duration of an instance of stuttering can be made much more apparent to both clinicians and researchers by the timing of instances of stuttering with stopwatches (and, of course, by instrumentally measuring the associated acoustic speech signal.) Although there can be no denying that stuttering duration varies within as well as between stutterers, it is still an empirical question whether differences in duration of stutterers *significantly* differentiate between stutterers for the purposes of either clinical or research endeavors (e.g., Schwartz & Conture, 1988 found duration of stuttering to be of minimal assistance when differentiating among subgroups of young stutterers). Interestingly, however, during therapy it has been our experience that perceived decreases in the duration of stuttering are one of the first aspects of stuttering to change as the child's fluency improves.

Severity of stuttering appears the least variable. Changes in severity, categorically measured as "mild, moderate, or severe," do occur as the problem worsens or improves but the rate of change is much slower. The relative stability of severity judgments probably relates to the fact that each category—mild, moderate, or severe—is sufficiently broad to contain a wide degree of variation. On the other hand, it is probably easier to objectify changes in the frequency of occurrence of all or particular types of disfluency because they can be more finely measured as a percentage and because of their inherent volatility.

Variations Within and Between Young Stutterers

Variations between young stutterers. One issue in the area of childhood stuttering that seems to have generated more heat than light is the discussion of whether there are significant differences between stutterers themselves (cf. St. Onge, 1963). Putting aside for the moment the various theoretical aspects of this discussion, researchers (Daly, 1981; Preus, 1982; Schwartz & Conture, 1988; Van Riper, 1971) are beginning to find evidence that childhood stuttering may not only arises from different origins but once begun the problem may develop along parallel but different routes. We hasten to add, however, that there is little information regarding whether these separate routes are equal in terms of the recovery from the problem or persistence into adulthood or whether the number and nature of these subgroups or clusters change with time. As Schwartz and Conture (1988) point out, one of the better tests of the long-term existence of particular subgroups of young stutterers would be a "longitudinal study whereby cluster analysis was performed on different samples of the same subjects collected at different times" (p. 69). In this way Schwartz and Conture (1988) suggest we would better understand whether "certain subjects might have shifted cluster membership (or whether) the nature and number of clusters themselves remained relatively intact" (p. 69).

Variations within young stutterers. Clinicians who manage stuttering in children must routinely try to decide whether the change in the child's stuttering during therapy is solely due to therapy or simply because of the properties of childhood stuttering. Perhaps individual variations in stuttering reflect variations in the number and type of speaking situations that young stutterers experience at home and elsewhere (e.g., child trying to verbally attract the attention of another child or verbally requesting an object possessed by another child and so forth; (cf. Davis, 1940). Thus, increases or decreases in a young stutterer's stutterings may be associated with changed in various cognitive, emotive, physical, and communicative events internal (e.g., fatigue, excitement, etc.) as well as external (e.g., parental demand for rapid, precise oral language) to the child. Furthermore, these associated events or states interact and vary rapidly as well as unpredictably. Thus, it is not particularly easy to predict, with any degree of precision, when, where, and how much any particular young stutterer will increase or decrease his or her stuttering frequency.

Static versus dynamic variation. Variations between young stutterers can, of course, also be *static* (relatively predictable or constant differences) or *dynamic* (relatively unpredictable, constantly changing differences). Furthermore, both static and dynamic variation can take one or both of two forms: (a) variations in the frequency, type, severity and duration of stuttering and (b) variations in related speech and nonspeech behaviors or attributes. Because most research efforts have chiefly been directed at uncovering differences between stutterers and normally fluent speakers, little is known regarding whether variations *within* and *between* young stutterers are static versus dynamic or most apt to be related to stuttered speech behavior versus other related behaviors.

When managing stuttering in children, clinicians must consider that youngsters who stutter differ between themselves in terms of frequency, duration, type, and severity of stuttering. Likewise, when researching stuttering in children, researchers must consider differences between young stutterers because it has been shown, for example, that differences among stutterers in terms of stuttering severity influence such diverse phenomena as reactions to delayed auditory feedback (cf. Bloodstein, 1987, p. 317) and initiation/execution times for manual and oral counting (e.g., Borden, 1983). Of course, it is possible that differences in other behaviors and characteristics, for

example, differences in diadochokinetic rate, may more readily account for differences in stutterers' reaction times and reactions to DAF than differences in their severity of stuttering. These, however, are empirical issues which await further study.

ROADBLOCKS TO RESEARCH

Forgetting That the Past is the Best Predictor of the Present and Future

The past is the potentially greatest roadblock to future research in stuttering, at least with regard to the definition of stuttering and categorization of stutterers in childhood. Previous publications in the area of stuttering are replete with reports where childhood stuttering and stutterers were reported on the basis of either vague, unspecified behavioral criteria or consensus judgments of two or three colleagues without explication of the behavioral criteria employed by the various judges. Likewise, this literature contains numerous publications in which the descriptors of stuttering and related behavior involve not only the person's behavior but the observer's guesstimation of the speaker's supposed intention or reason for exhibiting the behavior, for example, "fillers," "starters," and "avoiders." If the past is one of the best predictors of the present and future, then unless change is made, it is unlikely that criterion- or norm-referenced, intention-free definitions of stuttering or stutterers will be developed. Perhaps, as Pogo said, "We have met the enemy and he is us."

Treating Subjective Impressions as Objective Data

There is clearly room for subjective impressions and perceptions in the field of stuttering. However, when they are considered equivalent to descriptions of behavior based on direct observations, there would appear to be cause for concern. For example, it is not unusual for clinicians and researchers alike to report the severity level of their clients or subjects without reporting the criteria used to arrive at these decisions. Mild, moderate, and severe stuttering is meaningless unless the quantitative and qualitative data used to arrive at these decisions are made apparent. At the least, scales like those of the Iowa test (Johnson, 1961) or Stuttering Severity Instrument (Riley, 1980) state behavioral criteria that need to be reached before considering a child a "mild," "moderate," or "severe" stutterer. Unfortunately, researchers all too often ignore these criteria and assume that one person's judgment of "mild" is the same as another's. Thus, two studies, both supposedly containing mainly "severe" stutterers, may indeed contain two groups of subjects that are alike in name only because the criteria underlying the diagnostic labels are not made apparent and may in fact be quite divergent.

Blurring the Distinction Between "Stuttering" and "Stutterer"

All too often, the distinction between stuttering and stutterer is blurred. We have noted that, for example, sometimes a child considered to be normally fluent can produce approximately the same number of sound/syllable repetitions as a child considered to be a young stutterer. Thus, as much as we would like absolute, precise cut-offs for deciding who is and who is not a stutterer, the behavioral overlap between the two populations makes this a difficult proposition. Instead, we must consider what is a tolerable "degree of error." That is, using any set of criteria, what is an acceptable margin of error or probability level that some of the children we call stutterers are or will become normally fluent speakers and vice versa. It can't be over emphasized that normally fluent children produce within-word disfluencies—not very many, but they do produce them.

Replicating Studies but Using Dissimilar Subject

Conture (1987) states that "researchers in the area of stuttering (should) attend to subject and behavioral detail as much as they do procedural and instrumental detail. . . . Until we can replicate ALL aspects of each other's research, the subject and behavioral parts as much as the rest of the method, we can continue to expect to find divergent and inconsistent findings" (p. 121). In other words, one very important reason that replication in the area of stuttering doesn't always succeed is that experimenters pay far less attention to what they are considering stuttering and who they consider stutterers than they do other methodological aspects of their studies. Researchers need to make explicit the decision rules that permitted them to include or exclude a child as a stutterer.

When these subjects and their behaviors are poorly, loosely, or unclearly defined and described it is small wonder that independent replication results in different findings. Only in recent years have experimenters begun to specify the frequency and type of disfluency of their stuttering subjects and the criteria by which they have been evaluated as "mild, moderate, or severe." Although it should be realized that these rather global categories of severity are less than precise descriptors of stuttering, their increased use in published papers reflects a movement in the right direction.

Specification of subjects and behaviors is particularly important with children because they are so rapidly and constantly developing in so many different ways that "careful objective matching of subjects is (essential) if replication is going to stand a chance of confirming previous findings" (Conture, 1987, p. 121). Conture (1987) further states that "results obtained from apparently identical studies of stutterers can be significantly influenced by differences in subjects studied. And, until researchers make such differences more apparent in their subject descriptions, their colleagues will continue to

find it difficult to sort out the numerous reasons for contradictory findings among studies" (p. 121).

Reporting An "Effect" That is Less Than the Judges' Measurement Error

Any study that measures stuttering frequency as a dependent variable, needs to make clear what is considered stuttering and how the judges agreed with themselves and each other in making these measurements. It is particularly important to know whether any "effect," for example, a decrease in stuttering frequency, is greater than the judge's measurement error. If stuttering decreases by 5% from a baseline to experimental condition, the first question should be: What is the magnitude of difference between observers' judgments of stuttering frequency from one time to the next when compared to the magnitude of the "effect?" It is highly possible that measurement error is greater than the so-called effect of the experimental condition!

Further, if every type of speech disfluency produced by a child is considered stuttering, then it is highly likely that results will differ from those where only within-word disfluencies were considered stuttered. Likewise, if the experimenter is trying to study selected acoustic or speech production events during instances of a child's stuttering but does not specify the number and nature of specific disfluency types assessed, then it is possible that another researcher who tries to replicate this work may obtain different results merely because he or she is examining different types of "stuttered" disfluency such as sound prolongations versus sound/syllable repetitions. For example, Conture, McCall, and Brewer (1977) and Conture, Schwartz, and Brewer (1985) have quite clearly shown that laryngeal articulatory adjustments differ a great deal depending on whether sound/syllable repetitions or sound prolongations are produced as well as the phonetic nature of the sound stuttered upon.

FUTURE DIRECTIONS

Realization That Different Subjects May Produce Different Results

Clinicians and researchers will more clearly explicate the decision rules that permitted them to include or exclude a subject. They will come to better appreciate that stutterers' speech characteristics—frequency, type, and severity of stuttering—need to be made explicit because descriptive or experimental results may systematically differ for different stutterers. For example, a researcher may find slower laryngeal reaction times produced by stutterers with higher frequency of stuttering. Because stuttering doesn't occur in a vacuum and can be associated, particularly in children, with a variety of other speech and language problems, for example, phonological difficulties (e.g., Louko, Edwards, & Conture, 1988; Nippold, 1990; St. Louis & Hinzman, 1988), experimenters will more routinely report whether they screened their subjects for such associated problems. They will do this because they will realize that children who stutter but who *do not* exhibit concomitant problems may have different etiologies (and symptomatologies) than those who *do* exhibit concomitant problems. Such pre-experimental differences between children who stutter may significantly influence experimental findings.

Realization That There Are Important Differences Between Children and Adults Who Stutter

Clinicians and researchers will increasingly realize that it is unrealistic to expect the same number and nature of speech and related behaviors in children as adults who stutter because such expectations disregard the influence of development, learning history, and experience. Simply put, we won't expect to observe the same number and nature of speech and nonspeech behaviors in a child with a 6-month of history of stuttering that we might in an adult with a history of 20 years of stuttering. Clinicians and researchers will increasingly understand how cautious one must be when extrapolating backward from adults to children who stutter or forward from the behavior of children to that of adults. Future research may show that young stutterers exhibit many, if not all, of the same behaviors as older stutterers but only less frequently. To draw an analogy, both children and adults use nouns, but the number and frequency of noun usage is quite different between the talker groups, particularly when we compare preschoolers to adults.

Use of Computers That Model Instances of Stuttering and Help Identify Which Children Are Stutterers

Humans ultimately decide what a stuttering is and who is a stutterer. However, in the future, humans may be helped to better understand the speech production and acoustic signal associated with stuttering by means of computer modelling. Furthermore, computers have been and will continue to be applied to the recognition of instances of stuttering and eventually to the identification of which children are and are not at risk for stuttering. Of course, there is serious danger in relying on any machine to do our thinking for us. Thus, the factual information programmed into the computer should be sufficiently broad and detailed enough to capture the central tendency as well as variations in stutterings and stutterers. Further, the resulting program should be flexible and capable of being highly interactive with the end-user, for example, a clinician. Such flexibility and interactiveness should permit the clinician or researcher to modify aspects of the program as circumstances dictate and should help them see that it is them and not the computer

program which must and should make the final decision whether a child's disfluency is stuttered or whether a child is a stutterer. Given these cautions, computer programs have potential for helping clinicians and researchers expand or augment rather than replace their judgment of childhood stuttering. Computer modelling of instances of stuttering and computer-assisted identification of stuttering is an exciting area and one that will receive a fair amount of attention in the years ahead.

Use of Consensus to Decide What Is Stuttering in Children and Which Children Do It

Having agreed to disagree for so long about childhood stuttering perhaps it is now time to agree to agree. To bring the study of stuttering in children more fully into the arena of behavioral science, some degree of consensus of what is and who is stuttering needs to be reached. Although at present and into the immediately viewable future humans will remain the final arbitrators of what is stuttering and which young speakers should be classified as stuttering, there is nothing that says that some reasonable consensus cannot be reached by these same humans. Consensus, of course, is not a cure-all. Some instances of childhood stuttering and youngsters who stutter will be inappropriately labeled and because of this some children may wind up, at least temporarily, receiving inappropriate services. However, objective guidelines should reduce such difficulties from their present level while of course not eradicating them completely. In the area of childhood stuttering, where so many have, for so long, agreed to disagree about this or that theory or therapy, it would seem to be about time that these same individuals began to agree what stuttering is during childhood and which children do it.

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REFERENCES

ADAMS, M. (1980). The young stutterer: Diagnosis, treatment and assessment of progress. *Seminars in Speech, Language and Hearing, 1*, 289–299.

AINSWORTH, S., & FRASER, J. (1988). *If your child stutters: A guide for parents.* (3rd ed.), Memphis, TN: Speech Foundation of America.

BLOODSTEIN, O. (1987). *Handbook on stuttering,* 4th ed. Chicago: The National Easter Seal Society.

BOEHMLER, R. (1958). Listener responses to non-fluencies. *Journal of Speech and Hearing Research, 1*, 132–141.

BORDEN, G. (1983). Initiation versus execution time during manual and oral counting by stutterers. *Journal of Speech and Hearing Research, 26*, 389–396.

BRUTTEN, G. (1982). *The Speech Situation Checklist for Children: A Discriminant Analysis.* Paper presented to The Annual Meeting of American Speech, Language, and Hearing Association, Toronto, Canada.

CANTWELL, D., & BAKER, L. (1985). Psychiatric and learning disorders in children with speech and language disorders: A descriptive analysis. *Advances in Learning and Behavioral Disabilities, 4*, 29–47.

CONTURE, E. (1982). *Stuttering* (1st ed.). Englewood Cliffs, NJ: Prentice-Hall.

CONTURE, E. (1987). Studying young stutterers' speech productions: A procedural challenge. In H. Peters & W. Hulstijn (Eds.), *Speech motor dynamics in stuttering.* Wien/New York: Springer-Verlag.

CONTURE, E. (1990). *Stuttering* (2nd ed.), Englewood Cliffs: Prentice-Hall.

CONTURE, E., & CARUSO, A. (1978). A review of the Stoker Probe Technique for diagnosis and treatment of stuttering in young children. *Journal of Fluency Disorders, 3*, 297–298.

CONTURE, E., & CARUSO, A. (1987). Assessment and diagnosis of childhood dysfluency. In L. Rustin, H. Purser, & D. Rowley (Eds.), *Progress in the treatment of fluency disorders.* London: Taylor & Francis.

CONTURE, E., COLTON, R., & GLEASON J. (1988). Selected temporal aspects of coordination during fluent speech of young stutterers. *Journal of Speech and Hearing Research, 31*, 640–653.

CONTURE, E., MCCALL, G., & BREWER, D. (1977). Laryngeal behavior during stuttering. *Journal of Speech and Hearing Research, 20*, 661–668.

CONTURE, E., & SCHWARTZ, H. (1984). Children who stutter: Diagnosis and remediation. *Communicative Disorders, 9*, (1).

CONTURE, E., SCHWARTZ, H., & BREWER, D. (1985). Laryngeal behavior during stuttering: A further study. *Journal of Speech and Hearing Research, 28*, 233–240.

CURLEE, R. (1981). Observer agreement on disfluency and stuttering. *Journal of Speech and Hearing Research, 24*, 595–600.

DAVIS, D. M. (1940). The Relation of Repetitions in the Speech of Young Children to Measures of Language Maturity and Situational Factors: Part II & III. *Journal of Speech and Hearing Disorders, 5*, 235–246.

DALY, D. (1981). Differentiation of stuttering subgroups with Van Riper's developmental tracks: A preliminary study. *Journal of the National Student Speech Hearing Association, 9*, 89–101.

FRANKEN, M. (1987). Perceptual and acoustic evaluation of stuttering therapy. In H. F. M. Peters and W. Hulstijn (Eds.), *Speech motor dynamics in stuttering.* Wien/New York: Springer Verlag.

FRANKEN, M. (1988). *An instrument for the perceptual and acoustical evaluation of stuttering therapy.* Paper presented to the American Speech, Hearing and Language Association Conference, Boston, MA.

GREGORY, H., & HILL, D. (1984). Stuttering therapy for children. In W. Perkins (Ed.), *Stuttering disorders.* New York: Thieme-Stratton.

GUITAR, B., & PETERS, T. (1980). *Stuttering: An integration of contemporary therapies.* Memphis, TN: Speech Foundation of America.

HOMANS, G. (1965). Group Factors in Worker Productivity. In H. Proshansky & B. Seidenberg (Eds.) *Basic studies in social psychology* (pp. 592–604). New York: Holt, Rinehart, & Winston.

HOWELL, P., HAMILTON, A., & KRYIACOUPOULOS, A. (1986). Automatic detection of repetitions and prolongation in stuttered speech. In *Speech Input/Output Techniques and Applications.* IEE, 252–256.

HOWELL, P., & VAUSE, L. (1984). Acoustic analysis and perception of vowels in stuttered speech. *Journal of Acoustical Society of America, 79*, 1571–1579.

HOWELL, P., WILLIAMS, M., & VAUSE, L. (1987). In H. Peters & W. Hulstijn (Eds.), *Speech motor dynamics in stuttering* (pp. 371–380). New York: Springer/Verlag Press.

INGHAM, R. (1985). Assessment of stuttering in children. In J. Gruss (Ed.), *Stuttering therapy: Prevention and early inter-*

vention. Memphis, TN: Speech Foundation of America.

JOHNSON, W., BOEHMLER, R., DAHLSTROM, W., DARLEY, F., GOODSTEIN, L., KOOLS, S., NEELEY, J., PRATHER, W., SHERMAN, D., THURMAN, C., TROTTER, W., & WILLIAMS, D. (1959). *The onset of stuttering*. Minneapolis, MN: University of Minnesota Press.

JOHNSON, W., DARLEY, F., & SPRIESTERSBACH, D. (1963). *Diagnostic methods in speech pathology*. New York: Harper & Row.

JOHNSON, W. (1946). *People in quandaries*. New York: Harper & Row.

JOHNSON, W. (1961). *Stuttering and what you can do about it*. Minneapolis, MN: University of Minnesota Press.

KELLY, E., & CONTURE, E. (1988). Acoustic and perceptual correlates of adult stutterers' typical and imitated stutterings. *Journal of Fluency Disorders, 13*, 233–252.

LOUKO, L., EDWARDS, M., & CONTURE, E. (1988). *Phonological characteristics of young stutterers and their normally fluent peers*. Paper presented at American Speech, Hearing and Language Association Conference, Boston, MA.

MARTIN, R., & HAROLDSON, S. (1981). Stuttering identification: Standard definition and moment of stuttering. *Journal of Speech and Hearing Research, 24*, 59–63.

NIPPOLD, M. (1990). Concominant speech and language disorders in stuttering children: A critique of the literature. *Journal of Speech and Hearing Disorders, 55*, 51-60.

PREUS, A. (1981). *Attempts at identifying subgroups of stutterers*. Oslo, Norway: University of Oslo Press.

PROSEK, R., & RUNYAN, C. (1982). Temporal characteristics related to the discrimination of stutterers' and nonstutterers' speech samples. *Journal of Speech and Hearing Research, 25*, 29–33.

PROSEK, R., & RUNYAN, C. (1983). Effects of segment and pause manipulations on the identification of treated stutterers. *Journal of Speech and Hearing Research, 26*, 510–516.

RILEY, G. (1980). *Stuttering Severity Instrument for Young Children*. Revised edition. Tigard, Oregon: C.C. Publications.

RILEY, G. (1981). *Stuttering Prediction Instrument for Young Children*. Tigard Oregon: C.C. Publications.

RILEY, G., & RILEY, J. (1979). A component model for diagnosing and treating children who stutter. *Journal of Fluency Disorders, 4*, 279–293.

ROBINSON, F. (1946). *Introduction to stuttering*. Englewood Cliffs, NJ: Prentice-Hall.

SANDER, E. (1961). Reliability of the Iowa Speech Disfluency Test. *Journal of Speech Hearing Disorders Monograph Supplement, 7*, 21–30.

ST. LOUIS, K., & HINZMAN, A. (1988). A descriptive study of speech, language, and hearing characteristics of school-age stutterers. *Journal of Fluency Disorders, 13*, 331-356.

ST. ONGE, K. (1963). The stuttering syndrome. *Journal of Speech and Hearing Research, 6*, 195–197.

SCHIAVETTI, N. (1975). Judgments of stuttering severity as a function of type and locus of disfluency. *Folia Phoniatrica, 27*, 26–37.

SCHIAVETTI, N., SACCO, P., METZ, D., & SITLER, R. (1983). Direct magnitude estimation and interval scaling of stuttering severity. *Journal of Speech and Hearing Research, 12*, 568–573.

SCHWARTZ, H., & CONTURE, E. (1988). Subgrouping young stutterers: Preliminary behavioral observations. *Journal of Speech and Hearing Research, 31*, 62–71.

SHEEHAN, J., & MARTYN, M. (1970). Stuttering and its disappearance. *Journal of Speech and Hearing Research, 13*, 279–289.

SHERMAN, D., & MCDERMOTT, R. (1958). Individual ratings of severity of moments of stuttering. *Journal of Speech Hearing Research, 1*, 61–67.

STARKWEATHER, C. (1987). *Fluency and stuttering*. Englewood Cliffs, NJ: Prentice-Hall.

STOCKER, B. (1976). *Stocker Probe Technique for the Diagnosis and Treatment of Stuttering in Young Children*. Tulsa, OK: Modern Education.

THOMPSON, J. (1983). *Assessment of fluency in school-age children*. Resource Guide. Danville, IL: Interstate.

VAN RIPER, C. (1971). *The nature of stuttering*. Englewood Cliffs, NJ: Prentice-Hall.

VAN RIPER, C. (1982). *The nature of stuttering*. (2nd ed.), Englewood Cliffs, NJ: Prentice-Hall.

WILLIAMS, D., & KENT, L. (1958). Listener Evaluations of Speech Interruptions. *Journal of Speech and Hearing Research, 1*, 124–131.

WILLIAMS, D., & SILVERMAN, F. (1968). Note concerning articulation of school-age stutterers. *Perceptual and Motor Skills, 27*, 713–714.

WINGATE, M. (1964). A standard definition of stuttering. *Journal of Speech and Hearing Disorders, 29*, 484–489.

YAIRI, E. (1981). Disfluencies of normally speaking two-year-old children. *Journal of Speech and Hearing Research, 24*, 490–495.

YAIRI, E. (1982). Longitudinal studies of disfluencies in two-year-old children. *Journal of Speech and Hearing Research, 25*, 155–160.

YAIRI, E., & LEWIS, B. (1984). Disfluencies at the onset of stuttering. *Journal of Speech and Hearing Research, 27*, 155–159.

YOUNG, M. (1975). Observer agreement for marking moments of stuttering. *Journal of Speech and Hearing Research, 18*, 530–540.

YOUNG, M. (1984). Identification of stuttering and stutterers. In R. Curlee & W. Perkins (Eds.), *Nature and treatment of stuttering: New directions*. San Diego, CA: College-Hill Press.

ZEBROWSKI, P., & CONTURE, E. (1989). Judgments of disfluency by mothers of young stutterers. *Journal of Speech and Hearing Research, 32*, 625–634.

Chapter 2

SUBJECT DEFINITION AND SELECTION CRITERIA FOR STUTTERING RESEARCH IN ADULT SUBJECTS

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The principal topics assigned to this author were to review subject definition and selection criteria reported in experiments investigating speech behaviors in adult stutterers, to determine whether the typical description of these criteria is sufficient to meet the demands of scientific investigation and replication, and to discuss a number of topics that should be considered in the development of subject definition and selection criteria. Although the organization of this paper is largely motivated by the assigned topics, I have deviated somewhat by reviewing in Section I the definitions of stutterers and stuttering that typically appear in the literature. The review is followed by a discussion of how the precise and careful use of these definitions bear directly on the development of subject definition and selection criteria. Section II represents the primary focus of the paper and includes: (a) a review of typical subject definition and selection criteria, and (b) a discussion of a number of topics that should be considered in the development of these criteria.

The argument that appropriate subject definition and selection criteria is an essential component of good experimental technique is based on the ubiquitous observations that so many of the overt characteristics of the disorder are highly variable across subjects at all levels of measurement. The argument developed here is that until the many facets of the heterogeneity of stuttering are better understood, criteria should err on the side of over-defining rather than under-defining essential details about the stutterer and his or her behaviors.

Finally, because this paper centers on experiments dealing with adults, much of what is written here presumes that the procedures employed in the experiments of interest are common to that population and are more physiologically based and invasive than those commonly used with children who stutter. Problems associated with the development of subject definition and selection criteria in the latter population group are considered in detail in a separate chapter entitled "Childhood stuttering: What is it and who does it?" by Edward Conture appearing in Chapter 1.

SECTION I: DEFINING STUTTERING AND STUTTERERS

I.A. An Overview

Definitions of stuttering continue to evolve with our theories and abilities to measure various aspects of the disorder. Traditionally, most definitions are descriptions of behaviors. They are typically presented as a comprehensive list of behaviors that are common to all stutterers and that differentiate stuttering from normal speech. An often cited example of a descriptive definition is given by Wingate: "The term *stuttering* means: I. (a) Disruption in the fluency of verbal expression, which is (b) characterized by involuntary, audible or silent, repetitions or prolongations in the utterance of short speech elements, namely: sounds, syllables, and words of one syllable ... II. Sometimes the disruptions are (e) accompanied by accessory activities involving the speech apparatus, related or unrelated body structures, or stereotyped speech utterances ... III. Also, there are not infrequently (f) indications or report of the presence of an emotional state, ranging from a general condition of 'excitement' or 'tension' to more specific emotions ... (g) The immediate source of stuttering is some incoordination expressed in the peripheral speech mechanisms; the ultimate cause is presently unknown and may be complex or compound" (Wingate, 1964). Most definitions include at least the following three descriptions of the verbal behavior associated with stuttering; involuntary, repetitions, and prolongations. For example, stuttering is defined in the International Classification of Diseases as "disorders in the rhythm of speech, in which the individual knows precisely what he wishes to say, but at the time is unable to say it because of an involuntary, repetitive prolongation or cessation of a sound" (World Health Organization, 1977, p. 202). For a good discussion of the various categories of definitions see Van Riper, 1982, Chapter 2.

I.B. Needs and Future Directions

I.B.1. Disfluency, dysfluency, and fluency. The intent prescribed to the terms *disfluency, dysfluency,* and *fluency* varies considerably as a function of the distinctions among theoretical models of stuttering. This issue is considered in sufficient detail elsewhere, for example, Ham (1989), Perkins (1984), and Wingate (1984a,b, 1988). There should be no disagreement, however, about the critical necessity to make explicit, especially for research purposes, the descriptions of the speech behavior(s) under observation. By way of example, I will throughout this paper retain distinct definitions for the terms *disfluency, dysfluency,* and *fluency,* largely following the rationale reported by Wingate (1984a,b). *Disfluency* is used here as a general referent, pertaining to the usual and normal disruptions in the patterns of speech movements that are perceived as "fluent speech." *Dysfluency,* on the other hand, is used to mean abnormal disruption in the normal patterns of speech movements. *Fluency* is used in the perceptual sense, to mean the realization of flowing, smooth, and easily produced speech; that is, as an abstraction of the underlying articulatory gestures. Thus, a sample of speech produced by a normal talker that has been judged to be fluent, as defined here, may include, at the level of speech production, disfluent segments but not dysfluent ones. Once again, the critical point is that these terms, and the criteria developed to operationally define them, must be made explicit. It will be shown in Section II that the failure to do so can lead to conflicting conclusions about a number of aspects related to stuttering.

I.B.2. Stutterer's self-identification and stuttering-identification. In defining stuttering and stutterers, infrequent attention seems to be given to the stutterer's "self-identification" and "stuttering-identification." In particular, the stutterer's identification of a dysfluency can be very important since, as discussed below in more detail, it is often the case that disagreement will occur in fluent-dysfluent judgments that are based on data representing different accessible levels of measurement, that is, perceptual, acoustic, movement, and neuromuscular. For example, it is not unusual that an utterance is judged "fluent" at the perceptual level by an experienced listener, while analysis at deeper levels of speech, kinematic for instance, indicate inappropriate or "dysfluent" production. Until the distinction between fluent, disfluent, and dysfluent speech is better understood, the adult stutterer's judgment in the classification of him- or herself as a stutterer and in the fluency-dysfluency distinction of his or her speech should be encouraged.

I.B.3. Voluntary and involuntary speech motor output. The distinction between "voluntary" and "involuntary" in defining disfluency and/or dysfluency is not consistently made, although it may represent a critical distinction between certain types of dysfluency exhibited by stutterers and disfluency exhibited by adults who do not stutter. The significance of "involuntary" in definitions of stuttering and stutterers is discussed in detail by Perkins (1983) in response to a review article on stuttering by Andrews and his colleagues (Andrews et al., 1983) and need not be elaborated in great detail here. By way of a brief example, Perkins (1983) writes that the presumption is that a voluntary disfluency in the adult population results from "linguistic uncertainty." This implies that a voluntary disfluency, as in the prolongation of the isolated vowel /a/ for example, is a voluntary strategy invoked by the speaker while attempting to resolve a high-level linguistic query, such as in lexical retrieval. On the hand, the mechanisms underlying involuntary speech acts are far less agreed upon. Citing Perkins (1983) once again as an example, dysfluency "presumably is a motor speech blockage." Certainly, one can argue with linguistic uncertainty and speech motor blockage models of the voluntary-involuntary distinction, but until the volition of speech motor output can be measured with validity, the stutterer's identification of his/her fluent and dysfluent speech should be encouraged. That is, stutterers appear to be in a better position to subjectively rate their utterances as involuntary dysfluent, voluntary disfluent, or fluent than are listener-judges.

These two notions, the stutterer's self-identification and the voluntary-involuntary distinction, are important because they appear to be essential parameters in distinguishing among disfluent, dysfluent, and perceptually fluent speech. The significance of developing criteria to explicitly differentiate among involuntary dysfluency, voluntary disfluency, and perceptual fluency will be discussed in greater detail in Section II.B.3.

I.B.4. Core versus secondary stuttering behaviors. Since many developmental models of stuttering consider some form of repetitions to represent the "core" of stuttering (e.g., Bloodstein, 1987; Stromsta, 1986; Van Riper, 1982) the inclusion of core behaviors in a definition appears warranted.[1] Beyond "involuntary repetitions," and perhaps somewhat secondarily the duration and frequency of prolongations and silent pauses, there appears to be less agreement as to what is sufficient to delimit stuttering, except perhaps for the frequent remark that defining stuttering is much more complicated than some would think. Of course, there is a wide variety of so-called secondary behaviors or accessory features associated with stuttering (e.g., Wingate, 1964), the specification of which would be important especially in the consideration of severity.

I.B.5. Inclusive definitions of stuttering. Although there currently seems to be a better appreciation for psychological effects on physiological behaviors (e.g., Smith & Weber, 1988; Zimmerman, 1980c) definitions for the most part are rarely inclusive of external and internal influences on the disorder. Rather, they are either predominantly psychologically or physiologically based. Comprehensive definitions of stuttering similar to the

[1]However chapter 3, of this volume, entitled "Research Procedures for Measuring Stuttering Severity" by Ludlow takes a different approach to the core versus secondary behavior distinction.

"integrated theory" notion proposed most recently by Smith and Weber (1988) need to be better developed.

Definitions of stuttering do not always include an amount of the suspected etiology of the disorder. For example, definitions could include the notion that involuntary "core" behaviors occur as a consequence of deficits, at various levels, in temporal programming (e.g., Caruso, Abbs, Gracco, 1988; Kent, 1984), spatial or movement programming (e.g., Zimmerman, 1980c), or both temporal and spatial programming (e.g., Alfonso et al., (1986a; 1987a,b,c).

With a proper definition of stuttering, the researcher is better able to define stuttering subjects. Section II.A. shows, however, that researchers generally provide little definition of the stuttering populations that serve in their experiments, one consequence of which is that it often makes it difficult to make appropriate comparisons among experiments.

SECTION II. SUBJECT DEFINITION AND SELECTION CRITERIA

II.A. An Overview

A review of the literature indicates that little detail is given in journal articles regarding either subject definition or selection criteria. It is more often the case that stuttering severity is reported, although the means by which the severity estimate is determined is highly variable across experiments. What follows are examples of subject selection criteria (and subject definition, if given) that have been reported in recent or frequently cited research papers. The aim is to demonstrate the variability among published descriptions of experimental subjects and the criteria employed to select and define them. The following citations are in alphabetical order.

Many reports of experiments in the contemporary literature provide little information at all. For example, Freeman and Ushijima (1978) state only that their subjects were mild-moderate or severe. No other details of subject selection or definition criteria are given. Guitar et al., (1988) reports the gender of the subjects, that they were all native speakers of the same language, and had never received treatment for stuttering. The first author subjectively judged the severity of the subjects. The criteria for estimating severity were not given. Although the motivation for the Martin and Haroldson (1988) study was to experimentally increase stuttering frequency, so that the definition of stuttering, the frequency of stuttering, and the severity criteria, are crucial in this type of study, very little detail is given. The procedure employed is difficult to ascertain. "In the control room, the experimenter monitored all sessions auditorily and depressed a handswitch each time the subject stuttered. Stuttering was defined in terms of *moments* or *instances* of stuttering and not in terms of specific disfluency types." McClean (1987) reports that "informal assessment of conversations with the stutterers (7 adult male) suggested that as a group their stuttering severity ranged from mild to severe." No other details of subject selection or definition criteria are given. Zimmerman (1980a,b) and Zimmerman and Hanley (1983) used subjects who were enrolled in speech and hearing clinics at the time of data collection. The specific type of therapy is not mentioned. No details of the severity criteria are given: "They ranged in severity from mild to severe as judged by a certified speech-language pathologist."

Other reports of experiments provide some detail regarding subject selection criteria, though the type and amount vary in considerable degree. For example, Alfonso and his colleagues (Alfonso et al., 1986a,b; 1987a,b,c; Kalinowski & Alfonso, 1987; Story & Alfonso, 1988; Watson & Alfonso, 1982, 1983, 1987) used subjective and objective criteria to identify stutterers and group them on the basis of stuttering severity. Objective evaluations of stuttering frequency and type were completed using a combination of procedures described in the Stuttering Interview (SI) (Ryan, 1974) and the Stuttering Severity Instrument (SSI) (Riley, 1972). Subjective judgments of stuttering severity were obtained from certified speech-language pathologists and from the experimental subjects. Additional criteria are developed if severity ratings differ markedly between reading and conversational speech samples, among objective and subjective criteria (e.g., Watson & Alfonso, 1987), or if inter-test reliability, as a function of time periods or the identity of the judges, was low (Kalinowski & Alfonso, 1987). Caruso et al., (1988) assessed stutterers' dysfluency during a conversational speech sample using two objective measures: 1), mean stuttering frequency (MSF), and 2), mean stuttering duration (MSD). Stuttering was defined as *sound/syllable repetitions* and *sound prolongations*. Conture et al. (1977) used MSF during conversation and oral reading. Stuttering severity was determined by use of the Iowa scale (Johnson, Darley, & Spriestersbach, 1963) based on the MSF. All of the subjects for Conture et al. (1985) were receiving therapy at a university speech and hearing clinic. MSF for sound/syllable repetition, sound prolongation, or within-word pause was used as a measure of severity. Metz et al. (1983) and Sacco et al. (1987) selected subjects enrolled in a residential stuttering treatment program. They used MSF based on sound and syllable repetitions, sound prolongations, and/or broken words produced during an unidentified reading sample. Severity was calculated using the SSI (Riley, 1972; 1980). The Shapiro (1980) experiment required that the locus of stuttering, type of dysfluency, and stuttering severity be reliably determined. Subjects read the Rainbow Passage five times and performed the Job and TAT Tasks (Johnson et al., 1963). Subjects were accepted if the interjudge agreement across four judges met criteria. Operational definitions of severity of stuttering were obtained using the Stuttering Severity Scale (Johnson et al., 1963), and estimates of the specific type and locus of stuttering were subjectively made from videotape viewing.

II.B. Needs and Future Directions

The section above indicates that it is usually the case that one would find little detail in a journal article regarding the subject definition and selection criteria employed in the experiment. Stuttering severity estimates are given more often than subject definition and selection criteria, although the means by which the severity estimate is arrived at is highly variable across experiments. In what follows, certain issues relevant to the development of subject definition and selection criteria are discussed in detail. The issues represent broad areas of concern, and should not be construed as suggested minimal criteria. Rather, criteria should be developed as a function of the nature of the experiment at hand. The goal that all researchers should share, however, is that adequate descriptions should be given in sufficient detail so that experiments can be replicated and/or results can be appropriately interpreted and compared among experiments.

II.B.1. What identification measures should be used? The common use of a standardized perceptual test such as the SSI would lead to an obvious advantage of direct comparison of subjects across different experiments. Other identification measures should be considered, and include: (a) familial history of stuttering,[2] (b) type(s) and duration(s) of therapies received, the distinctive characteristics of a therapy program, a description of clinical goals (e.g., slowed speech rate, gentle onsets) of these therapies, and how well they are maintained in habitual speech, (c) estimates of covert stuttering severity, for example, the stutterer's judgment of the frequency and severity of dysfluency, secondary characteristics (e.g., Riley, 1972; Van Riper, 1982), descriptions of contextual conditions in which fluency and dysfluency are enhanced, and an estimate of the success to which the subject is able to use fluency enhancers to promote fluent speech, (d) estimates of overt stuttering severity, for example, the frequency and duration of repetitions, prolongations, and silent pauses, and (e) a description of the subject's fluent speech, for example, rate and naturalness. Speech samples gathered as part of the evaluation should be obtained during both extemporaneous and read speech, and the differentiating stuttering behaviors should be noted. Speech samples gathered as part of the evaluation and experimental run should routinely be video-taped.

However, perceptual testing alone poses a number of problems. The first is that the magnitude of stuttering severity is highly variable within subjects so that a classification of "severe" could be given to a subject tested in the morning while a classification of "moderate" could be given to the same subject tested in the afternoon (This problem is discussed in greater detail in Section II.B.4.) A second problem, if one accepts the notion of physiological loci, is that available standardized tests do not adequately differentiate between, for example, labial stutterers and laryngeal stutterers. The latter problem is related to a more serious shortcoming of subject selection and classification schemes made on the basis of standardized perceptual testing alone, in that no physiological measurement of speech is attempted. The omission of physiological description is a serious omission because: (a) a physiologically based classification may be more stable across time than a perceptual method, especially as the linguistic structure of dysfluent speech is better understood and the corresponding linguistic-physiological data base is increased, and (b) a dysfluent utterance may not be identified at the perceptual level. That is, while a segment of a stutterer's speech may be judged "fluent" by a group of listeners, the acoustic, and/or movement, and/or electromyographic signals underlying the perceptual segment may appear inappropriate or "dysfluent" (e.g., Alfonso et al., 1984; Baer & Alfonso, 1984; Shapiro, 1980).

A relatively noninvasive physiological-based battery could be devised. The following are examples of possible criteria that could be included in a subject selection and definition battery. Of course, the selection and applicability of physiological criteria are dependent upon the nature of the proposed experiment, the point, however, is that frequent and systematic use of certain physiological measures would enhance the validity of data comparison across different experiments. Noninvasive respiratory criteria, based on data obtained by a Respitrace inductive plethysmograph for example, could include the magnitude of the lung volume exchange for speech inspiration and expiration, or flow rates during fluent speech, because both have been shown to differentiate stutterers from nonstuttering adults, and, perhaps distinguish mild from severe stutterers (e.g., Lewis, 1975; Peters & Boves, 1987, 1988; Watson & Alfonso, 1987). An extensive body of literature on the differences between stutterers' and control subjects' acoustic reaction-time responses (e.g., Watson & Alfonso, 1983, 1987) suggest that predominantly laryngeal (and secondarily respiratory and supralaryngeal) noninvasive criteria could include acoustic reaction-time latencies for steady-state vowel responses. More direct noninvasive laryngeal estimates could be made using an electroglottograph with the reaction-time paradigm. Vocal-tract criteria based on direct measures of supralaryngeal articulator movements would require more technologically sophisticated noninvasive paradigms than those discussed above, and could include, for example, strain-gauge or opto-electronic movement transducers to measure lip and jaw displacement. These techniques are becoming more common in many laboratories. It would be important that supralaryngeal physiological criteria for the selection and definition of stuttering subjects be centered on organizational principles of speech motor control, that is, centered on good representatives of what are believed to be speech motor control parameters. For example, noninvasive lip and jaw displacement amplitude data could be analyzed to assess motor equiva-

[2]For a discussion of the problems associated with gathering familial history data, see the chapter entitled "A Review of the Evidence for Genetic Factors in Stuttering" by Pauls appearing elsewhere in this volume.

lence covariation and sequential ordering in labial gestures (Alfonso et al., 1986a, 1987b,c; Caruso et al., 1988). This issue is discussed in greater detail in Section II.B.3. It is less important to know and base group comparisons on displacement amplitudes and velocities of individual lip and jaw movements, and at what rates they move, than it is to know about the organizational principles underlying lip-jaw movements because (a) efficient, rapid, and fluent speech requires a relatively high degree of spatial and temporal coordination among the supralaryngeal speech structures, and (b) interspeaker variability at the motor control level is inherently less variable than at the phonetic level. Of course, data obtained by a combination of these techniques could be used to measure intersystem physiological parameters, for example, respiratory-laryngeal timing (e.g., Peters & Boves, 1988; Watson & Alfonso, 1987).

II.B.2. What behaviors during the moment of stuttering should be included in the definition? The type, duration, and severity[3] of the involuntary dysfluency should be identified during the moment of stuttering. Because the relationship between linguistic structure and speech motor specification in dysfluent speech is not fully understood, a description of the linguistic context in which the dysfluency occurred should be given. At the very least, the intended fluent phonetic target should be identified, for example, stressed syllable initial voiceless aspirated stop. Other linguistic descriptors that could be specified depending on the focus of the experiment include word position and content versus function word. A detailed consideration of psycholinguistic variables associated with stuttering is given in Wingate (1988). A number of secondary characteristics, including anxiety and emotional stress, should also be considered.

Although Shapiro (1980) concluded on analysis of EMG data that the perceptions of the judges as well as the subjects themselves regarding the identification of labial, laryngeal, and lingual predominate locus were erroneous, it might still be clinically and theoretically useful, particularly in regard to the subject's perception of his/her production of the dysfluency, to include in the definition a statement about clinician and/or client estimate of locus. We need more physiological data, in parallel with perceptual and acoustic, to determine with certainty whether or not stuttering results from a physiological disruption at one location, the larynx for example, or if the entire speech motor system fails simultaneously. Shapiro concludes that the stuttering may not be able to identify the location of the disruption, however, we don't have enough data to know this for certain, nor do we have enough data to know whether a breakdown at the larnyx, for example, occurs first and leads to failures in other components of the speech system in response to the laryngeal failure.

[3]For a detailed discussion of the problems associated with severity estimates see Ludlow chapter appearing elsewhere in this volume.

Finally, for those experiments based on physiological data it may be useful to segment the events surrounding a dysfluency. By way of example, Alfonso and Seider (1986) examined acoustic, respiratory and laryngeal kinematic data, and laryngeal electromyographic (EMG) data during an interval beginning with the termination of fluency, followed by an inaudible dysfluent period, followed by an audible dysfluent period, and ending with a fluent period. Laryngeal EMG and movement data showed that the laryngeal configuration was clearly different during the inaudible than during the audible periods of the dysfluent episode. The configuration appeared most inappropriate during the initial inaudible period and less so during the following audible period. Segmentation also allows for the comparison of speech motor events during the dysfluent periods with events immediately following, that is, during fluent production of the phonetic target. Thus, statements about the configuration of the vocal tract during moments of stuttering can be made in reference to the vocal tract configuration during more fluent episodes immediately preceding the dysfluency, and in reference to the vocal tract configuration immediately following the dysfluency and associated with the intended fluent phonetic target.

II.B.3. Is there such a thing as "normal disfluency" and can it be reliably differentiated from stuttering? Perceptual fluency spans a wide continuum, the endpoints of which could be identified as "severely fluent" to "severely dysfluent." There is large variability within the "normal" subsection of the continuum, ranging from something like "severely fluent" to "normal disfluent" and apparently even more variability within the "abnormal" subsection of the continuum, ranging from something like "abnormal fluent" to "severely dysfluent." Because the "normal" and "abnormal" subsections of the continuum appear to overlap perceptually, it seems that defining *abnormal fluency*, particularly when produced by severe stutterers, may be more straightforward than defining *normal disfluency*. That is, it may be more experimentally viable and more fruitful in the long run to modify the above question and to ask: is the perceptual "fluent" speech of severe stutterers similar to the fluent speech of control subjects? The latter form of the question may be more experimentally viable because: (a) the resolution involves extreme contrasts, the continuum endpoints, represented by a severe stutterer contrasted with a fluent control subject in the example here. The extreme contrast should be easier to differentiate than more subtle contrasts; for example, certain physiological characteristics of the perceptually fluent speech of a severe stutterer compared with the corresponding characteristics of a normal subject's fluent speech should be easier to differentiate than a mild stutterer's fluent speech compared with a normal subject's fluent speech, and (b) it would be easier to define a severely dysfluent stutterer than it would be to define a severely fluent control subject. The latter form of the question may be more fruitful because a better understanding of stutterers' perceptually fluent speech would represent a relatively direct and immediate increase in our understanding of

stutterers' speech motor control. Of course, in the long run we will need to understand better the fluency variability in the normal population as well as the stuttering population. Thus, the following discussion is a modification of the originally assigned question and asks: (a) can normal disfluency be reliably differentiated from stutterers' dysfluency, and (b) can stutterers' perceptually fluent speech be reliably differentiated from normal fluent speech?

Considering normal disfluency first, certainly adults who do not stutter do repeat and prolong a variety of speech segments, usually words and phrases, and interject pauses between words and phrases. Generally, the speech segments in which repetitions occur differentiate the groups; nonstutterers predominantly repeat whole words and phrases whereas stutterers predominantly repeat sounds and syllables. The duration of pauses and prolongations may also distinguish normal disfluency from abnormal dysfluency. However, the frequency of repetitions and the duration of prolongations, regardless of the speech segment in which these occur, are far less in magnitude in the nonstuttering population than in the stuttering population. That is, stutterers do more of everything; repetitions, prolongations, and pauses. Normal disfluencies are not usually accompanied by secondary characteristics and may be less susceptible to higher linguistic influences, for example, word order and word type (see Starkweather, 1987, Chapter 5 for a more detailed discussion of normal disfluency). An important assumption underlying the distinction between normal and abnormal speech is that normal speech movements are voluntary whereas stutterers' dysfluencies are not. Although it may be difficult, and perhaps impossible, to ascertain the volition of speech motor output, determining the extent to which repetitions, prolongations, and pauses are under voluntary motor control may be the ultimate test of the normal disfluency versus abnormal dysfluency distinction.

Considering stutterers' perceptually fluent speech next, there is considerably more data regarding the contrast between stutterers' perceptually fluent speech and normal fluent speech than in the contrast discussed above. However, some experimenters conclude that stutterers' perceptually fluent speech is not different than control subjects' fluent speech, while the majority of experimenters seem to think that it is (see, for example, Van Riper, 1982, Chapter 16; Bloodstein, 1987, Chapter 1). Rather than review a relatively large literature here, it may be more useful in regards to the development of subject definition and selection criteria to discuss a few of the reasons underlying the conflicts in the results of these types of experiments. It should be noted first, however, that the conflicting results are no doubt confounded by the lack of adequate definitions and criteria for distinguishing among certain essential characteristics of stuttering discussed in Section I, for example, voluntary disfluency, involuntary dysfluency, and perceptual fluency. That is, the results of two experiments may generate conflicting conclusions whether or not stutterers' perceptually fluent speech is similar to normally fluent speech simply because the criteria (which may or may not be reported) for distinguishing the stutterers' perceptually fluent speech from their dysfluent speech differed across the two experiments.

Second, the majority of experiments are based on perceptual data alone, some are based on perceptual and speech acoustic data, and far less include kinematic and neuromuscular data in parallel with perceptual and acoustic. One source of the conflict was discussed above in Section II.B.1, that is, while a segment of a stutterer's speech may appear normal or "fluent" at the perceptual level, it may appear abnormal or "dysfluent" at the acoustic, and/or movement and muscular levels (Alfonso et al., 1984; Baer & Alfonso, 1984; Shapiro, 1980). Thus, a comparison based on perceptual data alone could indicate no difference between the groups, while a comparison of the same utterances using the same perceptual criteria for fluency and dysfluency but based on physiological data could find significant group differences. Clearly, perceptual data alone are too far removed from the source to be able to make detailed analyses of the fluent-dysfluent distinction, and as such would mark only the most obvious instances of stuttering. The lack of physiological data addressing this issue is of concern for other reasons. For example, the question of determining whether the perceptually "fluent" speech of severe stutterers is similar to the fluent speech of control subjects is important because the answer to the question will help determine whether the stutterers' speech motor system exhibits generalized spatio-temporal abnormalities regardless of the perceived fluency, or whether, alternatively, it behaves normally except during moments of dysfluency. It is generally the case that perceptual and acoustic data in the absence of simultaneously gathered physiological data are not sufficient to answer questions about speech motor control.

A second reason for the conflict in the results of reported literature on the distinction between stutterers' perceptually fluent speech and nonstutterers fluent speech is that the routinely posed form of the question, "Is stutterers' fluent speech similar to the fluent speech of adults who do not stutter," is too broad. It is possible that certain aspects of speech do not differentiate the groups while other aspects do differentiate the groups. For example, Baken et al., (1983) found no differences between stutterers and control subjects in chest wall preposturing maneuvers immediately preceding fluent speech. Yet, significant differences in subglottic pressure (e.g., Lewis, 1975), flow rates (e.g., Hutchinson, 1975), and lung volume charge and deflation for speech (e.g., Watson & Alfonso, 1987; Story, 1990) have been observed between the groups. Thus, it is less appropriate to ask whether or not respiratory control, per se, (and certainly not speech production, in general), can be differentiated between the groups. Rather, it might be that stutterers and normal speakers perform certain speech respiratory gestures in a similar fashion, namely respiratory preposturing, but that other aspects of speech respiration, namely the magnitude of the inspiratory charge, are performed differentially.

A third and perhaps most important reason for the conflict in the results of these types of experiments is that group comparisons are frequently based on physiological data that reflect relatively variant phonetic level speech gestures. Group data based on phonetic level contrasts are inherently unstable because spatial and temporal control of the speech structures to mark phonetic distinctions varies as a function of phonetic context, stress and rate, dialect, and individual speaker preferences. Rather, group comparisons should be based on relatively stable spatial and temporal characteristics of normal speech dynamics, for example those that best reflect organizational principles of speech motor control. Dynamic parameters that are relatively invariant across multiple productions of an utterance are thought to be good representatives of speech motor control parameters (e.g., Gracco & Abbs, 1986). Thus, an appropriate comparison would be one based on the extent to which the magnitude of the relatively stable spatial and temporal characteristics of normal speech dynamics approximates corresponding characteristics of stutterers' perceptually fluent speech (Alfonso et al., 1986a, 1987b,c; Caruso et al., 1988). For example, motor equivalence covariation for speech is based on the observation that normal subjects control the relative displacement of individual articulators (e.g., the tongue and jaw) enlisted in a vocal tract gesture (e.g., alveolar closure) in such a way that the variability of the gesture is less than the variability associated with the individual articulators comprising the gesture. Accordingly, it is less appropriate to base group comparison on displacement amplitude and peak velocity of a single articulator, the tongue or the jaw for example, than it would be to compare the relative organization of the combined tongue-jaw displacement, because the latter comparison reflects a relatively stable component of speech dynamics while the former reflects idiosyncratic speaker preference. To summarize, the question of whether stutterers' perceptually fluent speech is similar to the fluent speech of nonstuttering adults can be appropriately addressed if criteria are developed that: (a) objectively distinguish among perceptually fluent, disfluent, and dysfluent utterances, (b) define the question more precisely, that is, provide more detail about specific aspects of speech production, (c) require appropriate types of data, and (d) require group comparisons based on appropriate speech behaviors.

II.B.4. How can a definition take into account variability in stuttering behavior? At least three types of variability should be taken into account. The first is associated with the well-known variability in the frequency and severity of dysfluent behaviors as a function of various external conditions. For example, dysfluent behaviors decrease and perceptually fluent behaviors increase with manipulation of auditory feedback; when the intensity of the feedback signal is increased, decreased, or masked by various techniques, and when the time of arrival of the auditory feedback signal to the talkers' ears is delayed. Fluency is enhanced by imposing an external rhythmical marker on the speech task, for example, a metronome signal, choral reading, and singing. Finally, fluency is enhanced by the adaptation effect. More of these conditions will enhance fluency in nonstuttering adults as well as stutterers. The exception seems to be the delayed auditory feedback (DAF) paradigm, where a delay in the signal could result in increased dysfluency in some nonstuttering adults, although normal speakers vary widely in their suspectibility to DAF (Alfonso, 1974). Thus, alterations of the feedback signal, imposition of a rhythmical marker, and multiple repetitions of the same speech task increase fluency in most talkers, stutterers and nonstutterers alike. It appears possible that all of these conditions enhance fluency through a similar mechanism, that is, they act to highlight the control of prosody and perhaps other temporal parameters of the speech motor plan.

Likewise, there are a number of external conditions that increase dysfluent behaviors and decrease perceptually fluent behaviors. It is well known that the contextual surround, for example, stressful situations and tasks, and specific listeners, will increase the frequency and severity of dysfluency. Certain linguistic conditions, such as long words, content versus function words, and words carrying high information loading, are known to promote dysfluency. Once again, these conditions are known to similarly affect adult nonstutterers, though not to the degree seen in stutterers. On the other hand, there are those adult nonstutterers (and apparently stutterers) who become more fluent in seemingly stressful situations. A likely causal agent for the increase in dysfluency in these conditions for both stutterers and adults who do not stutter is the effect of stress on the speech motor system (e.g., Zimmerman, 1980c).

The second type of variability is associated with changes in the type, frequency, and/or severity of dysfluent behaviors that occur across varying units of time. As an example of the variation in dysfluent behaviors that occur across the long term, it is generally believed that very young children who stutter do so primarily by repetition and prolongation, while over the course of 2 to 4 years develop a variety of other overt behaviors not present at the onset. Thus, stuttering in adults is comparatively idiosyncratic and as such varies considerably across the adult stuttering population. There are other examples of long-term changes in dysfluent behaviors, although the basis for the change is not sufficiently understood. With respect to the example given here, the source and extent of the variability of "core" behaviors, the repetitions and prolongations in very young children, compared to the variability of "secondary" behaviors in older children and adults apparently remains unresolved (Stromsta, 1986; Van Riper, 1982). Of course, variations in dysfluent behaviors occur across much smaller units of time. In fact, the perception of stuttering frequency, severity, and type within the same subject can change significantly as a function of many of the external conditions mentioned above (e.g., stressful situations and tasks) over the course of hours and days. Thus, as was pointed out in Section II.B.1., the same subject can be classified as a mild-moderate stutterer in a morning session and using the same severity rating instrument be classified as

a severe-moderate stutterer in an afternoon session. This poses a particular problem in the development of subject definition and selection criteria and illustrates the importance of accounting for the variety of conditions that are known to induce short- and long-term fluency variations.

A third type of variability that could be included in subject definition and selection criteria is the type observed at the speech motor level. Greater variability for stutterers compared to control subjects at every accessible level of speech production, namely, acoustic, movement, and neuromuscular, is frequently reported and must be considered a robust observation. Relatively high levels of variability can, of course, be of serious consequences in meeting the demands of rapid, fluent speech. High variability implies that stutterers' control of the movements of the speech structures is less precise than adults who do not stutter. The lack of precision implies that stutterers lack the flexibility observed in normal speech, the flexibility that forms the basis for co-production of speech segments and in other human multiarticulate systems. Thus, the increased variability observed in the kinematics of many structures of the speech mechanism, and more importantly, the increased variability associated with various organizational components of fluent speech (e.g., the tongue-jaw synergies referred to in Section II.B.3.) suggest that stutterers' control of the speech motor system lacks the flexibility and efficiency to meet the demands of rapid, fluent speech (e.g., Alfonso et al., 1985, 1987c; Stromsta, 1986).

In summary, subject definition and selection criteria should account for the variability that is frequently observed at all levels of stuttering behaviors. In regards to treatment of the data, descriptions of data dispersion in addition to central tendency should be reported. Reporting the complete data base, perhaps as an appendix, would often be appropriate. Statements regarding the suspected source or the conditions promoting the variability, and the effect of the source or conditions on the variability, should be included; for example, fluency enhancement by rhythmical stimulation, dysfluency increase by psychological and physiological stress, accompanied by a statement regarding the magnitude of the fluency change. Finally, we need to understand better the relationship between the magnitude of the variability and stuttering severity.

II.B.5. What are the commonalities among stutterers? There are certain characteristics that appear to be common among stutterers as a group and should be considered in developing subject definition and selection criteria. For example, all stutterers seem to experience the notion of stutterer-identification discussed above in Section I.B.2. That is, regardless of severity, or the degree to which overt or covert manifestations of stuttering are realized, stutterers seem to know that they are stutterers. If this is indeed true, then the notion of self-identification should be a central component of the definition of stuttering and stutterers. The stutterers' identification of themselves as stutterers and their identification of their moments of stuttering should focus the development of perceptual and physiological criteria.

Based on family history data, many researchers have concluded that the predisposition to stutter is genetically transmitted. For example, Van Riper (1982) concludes: "This incidence is so much greater than that found generally in the population (about 5%) that it is difficult to believe that environmental factors alone could account for the results" (p. 330). Family history studies show that more males than females stutter, that there is a high incidence of stuttering within families, and that the risk of stuttering is higher if the mother, rather than the father, stutters. Certain of these studies allow the determination of risk to relatives; stuttering occurred in about 20% of male relatives and about 5% of female relatives of male stutterers, whereas stuttering occurred in about 25% of male relatives and 12% of female relatives of female stutterers (Kidd et al., 1978, 1981). However, as Pauls points out in Chapter 4 appearing in this volume entitled "A Review of the Evidence for Genetic Factors in Stuttering," there are significant limitations to the family study method. Coupled with the fact that these limitations make it impossible to understand the exact nature of the transmission, and that a disorder as common as stuttering is almost certain to be etiologically and genetically heterogeneous, the inclusion of family history data in subject definition and selection criteria should be cautiously considered.

The onset of stuttering is fairly common across stutterers. Most stuttering develops during childhood and is usually marked initially by syllable repetitions and prolongations. Adult stutterers who demonstrate a characteristic childhood onset should be distinguished from the relatively few adult stutterers who report sudden onset later in life.

Finally, certain physiological characteristics of the speech motor system are shared by most stutterers. For example, the stutterers' speech motor system is inappropriately susceptible to psychological and physiological stress. Fluency, measured at the output of the system primarily by perceptual criteria, is enhanced by rhythmical stimulation. The reaction-time of the stutterers' speech motor system is slower than normal speakers. And the stutterers' system may be characterized by a disability in spatial, temporal, or spatio-temporal condition, particularly of the type discussed in Section II.B.3.

II.B.6. What would be the benefits to research in having a consensus definition? While it is likely that reaching the ultimate consensus definition is not possible to achieve, the alternative, that is to continue without suggestive guidelines would lead to a continuation of the variability in subject definition and selection criteria illustrated in Section II.A. One of the aims of this paper is to demonstrate the different ways in which current definitions and criteria are inadequate. With respect to definitions of stuttering, there are many instances where data have been pooled across such factors as severity and type so that a clear interpretation of the results is not possible. The lack of clearly stated subject selection criteria may have even more serious consequence on data interpretation because of the heterogeneous nature of the disorder. For example, data are frequently pooled across such

variables as subject severity, varying clinical treatment influences on perceptually fluent speech patterns, and idiosyncratic dysfluent verbal and nonverbal behaviors.

There are, however, obvious and significant limitations to the consensus approach. The first is related to the wide varieties of stuttering behaviors and subjects that are often examined. Overly precise definitions may exclude critical members of a category while overly general definitions would not make the necessary distinctions between members of a category. Secondly, standardized subject definition and selection criteria would be in a constant state of reassessment as our knowledge of the disorder continues to increase.

Many of the benefits of a consensus definition can be achieved by the routine practice of reporting sufficient details of the experiment so that, at the very least, the experiment can adequately be replicated. The precise details will vary as a function of the nature of the experiment, yet it would be difficult to imagine many cases where basic descriptors, for example, severity ratings of the dysfluencies and subjects under examination, could be omitted. The paper presented here, as well as others appearing in this volume, discuss a number of factors that bear on subject definition and selection criteria. Undoubtedly, other factors not listed here could also be included. Thus, the consensus opinion should be that subject definition and selection criteria adequately deal with the heterogeneity of stuttering, that they be specifically generated as a function of the experiment at hand, and that until the significance of the heterogeneity is better understood, definitions and criteria should err on the side of over- rather than under-defining critical details of the experiment. Included in the notion of over-defining in the context of a heterogeneous behavior is the treatment of the data. Because the reported data are often used by future researchers in a way not related to the aim of the original experiment, it would be appropriate to publish certain forms of individual subject data as an appendix, in addition to the typical reporting of averaged data, especially when the data represent physiological estimates of stuttering that are technically difficult to collect and/or potentially hazardous to the subject. The consensus, then, becomes one of intent rather than the specification of consensus criteria.

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REFERENCES

ALFONSO, P. J. (1974). *Differentiating phonemic and spectrographic speech characteristics of DAF susceptibility.* Unpublished Master's thesis, Western Michigan University.

ALFONSO, P. J., WATSON, B. C., & BAER, T. (1984). Muscle, movement, and acoustic measurements of stutterers' laryngeal reaction times. In M. Edwards (Ed.), *Proceedings of the 19th Congress of the International Association of Logopedics and Phoniatrics* (Vol. II, pp. 580–585). Perth, Scotland: Danscott Print Limited.

ALFONSO, P. J., WATSON, B. C., BAER, T., SAWASHIMA, M., HIROSE, H., KIRITANI, S., NIIMI, S., ITOH, K., SEKIMOTO, S., HONDA, K., & IMAGAWA, H. (1985). The organization of supralaryngeal articulation in stutterers' fluent speech production: A preliminary report. *Annual Bulletin of the Research Institute of Logopedics and Phoniatrics, 19,* 191–200.

ALFONSO, P. J., WATSON, B. C., & SEIDER, R. A. (1986a). Tongue and jaw articulation in stutterers' fluent speech. *Asha,* 28–10, 166.

ALFONSO, P. J., & SEIDER, R. A. (1986b). Laryngeal and respiratory physiological characteristics of inaudible and audible dysfluent production. In S. Hibi, D. Bless, M. Hirano (Eds.), *Proceedings of the International Congress on Voice.* Kurume, Japan: Kurume University, 1, 13–22.

ALFONSO, P. J., WATSON, B. C., & BAER, T. (1987a). Measuring stutterers' dynamical vocal-tract characteristics by x-ray microbeam pellet-tracking. In H. F. M. Peters and W. Hulstijn (Eds.), *Speech motor dynamics in stuttering* (pp. 141–150). Wien/New York: Springer-Verlag Press.

ALFONSO, P. J., STORY, R. S., & WATSON, B. C. (1987b). The organization of supralaryngeal articulation in stutterers' fluent speech production: A second report. *Annual Bulletin of the Research Institute of Logopedics and Phoniatrics, 21,* 117–129.

ALFONSO, P. J., STORY, R. S., & WATSON, B. C. (1987c). Spatial-temporal variability of tongue-jaw movements in stutterers' fluent speech. *Asha,* 29–10, 159.

ANDREWS, G., CRAIG, A., FEYER, A. M., HODDINOTT, S., HOWIE, P., & NEILSON, M. (1983). Stuttering: A review of research findings and theories circa 1982. *Journal of Speech and Hearing Disorders, 48,* 226–246.

BAER, T., & ALFONSO, P. J. (1984). On simultaneous neuromuscular, movement, and acoustic measures of speech articulation. In R. G. Daniloff (Ed.), *Articulation assessment and treatment issues.* San Diego: College Hill Press.

BAKEN, R. J., MCMANUS, D. A., & CAVALLO, S. A. (1983). Prephonatory chest wall posturing in stuttering. *Journal of Speech and Hearing Research, 26,* 444–450.

BLOODSTEIN, O. (1987). A handbook on stuttering. Chicago, IL: The National Easter Seal Society.

CARUSO, A. J., ABBS, J. H., & GRACCO, V. L. (1988). Kinematic analysis of multiple movement coordination during speech in stutterers. *Brain, 111,* 439–455.

CONTURE, E., MCCALL, G., & BREWER, D. (1977). Laryngeal behavior during stuttering. *Journal of Speech and Hearing Research, 20,* 661–668.

CONTURE, E., SCHWARTZ, H. D., BREWER, D. (1985). Laryngeal behavior during stuttering: A further study. *Journal of Speech and Hearing Research, 28,* 233–240.

FAIRBANKS, G. (1960). Voice and articulation drillbook. New York: Harper & Row.

FREEMAN, F. J., & USHIJIMA, T. (1978). Laryngeal muscle activity during stuttering. *Journal of Speech and Hearing Research, 21,* 538–562.

GRACCO, V. L., & ABBS, J. H. (1986). Variant and invariant characteristics of speech movements. *Experimental Brain Research, 65,* 156–166.

GUITAR, B., GUITAR, C., NEILSON, P., O'DWYER, N., ANDREWS, G. (1988). Onset sequencing of selected lip muscles in stutterers and nonstutterers. *Journal of Speech and Hearing Research, 31,* 28–35.

HAM, R. E. (1989). What are we measuring? *Journal of Fluency Disorders, 14,* 231–243.

HUTCHINSON, J. (1975). Aerodynamic patterns of stuttered speech. In L. M. Webster, & L. C. First (Eds.), *Vocal tract dynamics and dysfluency.* New York: Speech and Hearing Institute.

JOHNSON, W., DARLEY, F., & SPRIESTERSBACH, D. (1963). Diagnostic methods in speech pathology. New York: Harper and Row.

KALINOWSKI, J. S., & ALFONSO, P. J. (1987). Improvement in laryngeal reaction time with improvement in fluency. *Asha*, 29–10, 124.

KENT, R. D. (1984). Stuttering as a deficiency in temporal programming. In W. H. Perkins & R. Curlee (Eds.), *Nature and treatment of stuttering:* New Directions. San Diego, College-Hill Press.

KIDD, K. K., KIDD, J. R., & RECORDS, M. A. (1978). The possible causes of the sex ratio in stuttering and its implications. *Journal of Fluency Disorders, 3*, 13–24.

KIDD, K. K., HEIMBUCH, R. C., & RECORDS, M. A. (1981). Vertical transmission of susceptibility to stuttering with sex-modified threshold. *Proceedings of the National Academy of Sciences, 78*, 606–610.

LEWIS, J. J. (1975). *An aerodynamic study of 'artificial' fluency in stutterers.* Unpublished doctoral dissertation, Purdue University.

MARTIN, R. R., & HAROLDSON, S. K. (1988). An experimental increase in stuttering frequency. *Journal of Speech and Hearing Research, 31*, 272–274.

METZ, D. E., SAMAR, V. J., & SACCO, P. R. (1983). Acoustic analysis of stutterers' fluent speech before and after therapy. *Journal of Speech and Hearing Research, 26*, 531–536.

MCCLEAN, M. D. (1987). Surface EMG recording of the perioral reflexes: Preliminary observations on stutterers and nonstutterers. *Journal of Speech and Hearing Research, 30*, 283–287.

PERKINS, W. H. (1983). The problem of definition: Commentary on 'stuttering'. *Journal of Speech and Hearing Disorders, 48*, 246–249.

PERKINS, W. H. (1984). Stuttering as a categorical event: Barking up the wrong tree—reply to Wingate. *Journal of Speech and Hearing Disorders, 49*, 431–433.

PETERS, H. F. M., & BOVES, L. (1987). Aerodynamic functions in fluent speech utterances of stutterers in different speech conditions. In H. F. M. Peters and W. Hulstijn (Eds.), *Speech motor dynamics in stuttering.* Wien: Springer-Verlag.

PETERS, H. F. M., & BOVES, L. (1988). Coordination of aerodynamic and phonatory processes in fluent speech utterances of stutterers. *Journal of Speech and Hearing Disorders, 31*, 352–361.

RILEY, G. (1972). A stuttering severity instrument for children and adults. *Journal of Speech and Hearing Disorders, 37*, 314–322.

RILEY, G. (1980). *Stuttering Severity Instrument.* Tigard, Oregon: CC Publications.

RYAN, B. (1974). *Programmed therapy for stuttering in children and adults.* Springfield, IL: Charles C. Thomas.

SACCO, P. R., & METZ, D. E. (1987). Changes in stutterers' fundamental frequency contours following therapy. *Journal of Fluency Disorders, 12*, 1–8.

SHAPIRO, A. I. (1980). An electromyographic analysis of the fluent and dysfluent utterances of several types of stutterers. *Journal of Fluency Disorders, 5*, 203–231.

SMITH, A., & WEBER, C. (1988). The need for an integrated perspective on stuttering. *Asha*, 30–32.

STARKWEATHER, C. W. (1987). *Fluency and stuttering.* Englewood Cliffs, NJ: Prentice-Hall.

STORY, R. S., & ALFONSO, P. J. (1988). *Changes in respiratory kinematics following an intensive stuttering therapy program. Asha*, 30–10, 192.

STORY, R. S. (1990). *Pre- and post-therapy comparison of respiratory, laryngeal, and supra laryngeal kinematics of stutterers' fluent speech.* Unpublished Ph. D. dissertation, University of Connecticut.

STROMSTA, C. (1986). *Elements of stuttering.* Oshtemo, Michigan: Atsmorts Publishing.

VAN RIPER, C. (1982). The nature of stuttering (2nd Ed.). Englewood Cliffs, NJ: Prentice-Hall.

WATSON, B. C., & ALFONSO, P. J. (1982). A comparison of LRT and VOT values between stutterers and nonstutterers. *Journal of Fluency Disorders, 7*, 219–241.

WATSON, B. C., & ALFONSO, P. J. (1983). Foreperiod and stuttering severity effects on acoustic laryngeal reaction time. *Journal of Fluency Disorders, 8*, 183–205.

WATSON, B. C., & ALFONSO, P. J. (1987). Physiological bases of acoustic LRT in nonstutterers, mild stutterers, and severe stutterers. *Journal of Speech and Hearing Research, 30*, 434–447.

WINGATE, M. E. (1964). A standard definition of stuttering. *Journal of Speech and Hearing Disorders.* 29, 484–489.

WINGATE, M. E. (1984a). Definition is the problem. *Journal of Speech and Hearing Disorders, 49*, 429–431.

WINGATE, M. E. (1984b). Fluency, disfluency, dysfluency and stuttering. *Journal of Disorders, 9*, 163–168.

WINGATE, M. E. (1988). *The structure of stuttering: A psycholinguistic analysis:* New York: Springer-Verlag.

WORLD HEALTH ORGANIZATION (1977). *Manual of the international statistical classification of diseases, injuries, and causes of death* (Vol. 1). Geneva: World Health Organization.

ZIMMERMAN, G. (1980a). Articulatory dynamics of fluent utterances of stutterers and nonstutterers. *Journal of Speech and Hearing Research*, 23, 95–107.

ZIMMERMAN, G. (1980b). Articulatory behaviors associated with stuttering: Cinefluorographic analysis. *Journal of Speech and Hearing Research*, 23, 108–121.

ZIMMERMAN, G. (1980c). Stuttering: A disorder of movement. *Journal of Speech and Hearing Research*, 23, 122–136.

ZIMMERMAN, G., & HANLEY, J. M. (1983). A cinefluorographic investigation of repeated fluent productions of stutterers in an adaptation procedure. *Journal of Speech and Hearing Research, 26*, 35–42.

B. MEASUREMENT OF STUTTERING SEVERITY

Chapter 3

RESEARCH PROCEDURES FOR MEASURING STUTTERING SEVERITY

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ROADBLOCKS

One of the major difficulties in stuttering research is the lack of a uniform approach to quantifying the disorder. No agreement is available on methods for comparing subjects. This is a problem not unique to stuttering research; the same difficulty has affected voice research and dysarthria. However, in the area of stuttering the problem is somewhat worse because an instrumentally based totally objective approach is not currently available. An automatic computer analysis system for detecting and quantifying disfluencies seems unlikely in the near future.

Stuttering also differs from voice and dysarthria because it is episodic and most salient in a communication setting. Controlling the speech context is most difficult in a communication situation and the usual methods of experimentally controlling context such as reading stimuli, repetition or sentence construction, will alter stuttering frequency and severity to varying degrees across subjects. That is, the task may differ from the degree of impairment a subject exhibits during communication; some subjects stutter more during oral reading while others more during communication. Because the degree of handicap a speech disorder imposes on a subject depends upon the degree to which it interferes with communication, content validity is more of a constraint in assessment of stuttering than in voice or dysarthria. The latter two are less episodic and although task behavior may differ from communication, this difference varies less across subjects.

Because instrumental procedures are not readily available, stuttering assessment is more dependent upon perceptual judgments of subjects' speech behavior by listeners/examiners during speech communication for measuring the disorder than in voice or dysarthria research. Hence, the focus is on what listeners should detect and how they rate its severity. Both of these are dependent upon some agreement on what is stuttering and which aspects of stuttering behavior represent its severity. The recommendation of procedures for measuring stuttering severity depends upon agreement among researchers on these two issues; something which is not readily forthcoming. However, without some metric for comparing subjects across studies or comparing treatment effects, research in this area will continue to be impeded.

The purpose of this paper is to evaluate the utility of currently available approaches for measuring stuttering severity across subjects and to recommend approaches which would be useful for comparing subjects across studies rather than simply saying that they are mild, moderate, and severe. Secondly, procedures are needed for evaluating treatment effects across settings. That is, some metric is needed for stuttering research which could be applied uniformly by different investigators when evaluating treatments so that the benefits of different treatments can be contrasted.

Valid and reliable measures of stuttering severity are needed for two research purposes: (a) to relate the severity of stuttering to other subject characteristics and (b) to evaluate the effects of treatment on the severity of subjects' stuttering. It would be advantageous if one measure could be used for both these purposes. Further, although certain aspects of stuttering differ between children and adults, measures of severity should be applicable for both populations. The purpose of this paper is to suggest measurement approaches which might meet these needs.

Disclaimers

Methods for measuring stuttering severity across subjects and between treatments will not necessarily aid in the diagnosis or analysis of stuttering behavior. These are different purposes and will need other approaches. Many investigators are interested in a detailed analysis of how subjects stutter and the types of behaviors they exhibit in different settings. The focus here was not to address this need. Similarly, procedures for determining whether a speech disorder is stuttering or an expressive aphasia is not being addressed here.

Stuttering is manifest not only in its overt manifestation but also both covertly and in life-style changes. The fear of stuttering is a common complaint. However, this is difficult to quantify objectively across subjects and is not

being addressed in this paper. Further, many stutterers have life-style changes that have occurred as a result of their fear of stuttering. They may avoid using the telephone, use writing rather than telephoning and be less social. These issues are not addressed in this paper. However, they are clearly important in the long-term measurement of treatment benefits and must be included. The focus here is on measuring the severity of the disorder as it is manifest in the communication setting. Although this is a limited objective, the lack of some metric for measuring severity has been an important roadblock in stuttering research.

Definition of Stuttering

Before measuring stuttering severity, there should be consensus on which behaviors are part of the disorder. The World Health Organization International Classification of Diseases defined stuttering as, "disorders in the rhythm of speech, in which the individual knows precisely what he wishes to say, but at the time is unable to say it because of an involuntary, repetitive prolongation or cessation of a sound." Another frequently cited definition by Wingate (1964) includes only silent and audible repetitions of sounds and syllables and silent and audible prolongations. Both definitions have included only sound and syllable repetitions and prolongations. These definitions imply that audible and silent sound/syllable repetitions and prolongation are necessary and sufficient to define stuttering phenomena and that other behaviors are the result of the subject's reaction to, or expectation of, the occurrence of sound and syllable repetitions and prolongations (Wingate, 1964; Ham, 1986; Riley, 1972; Stromstra, 1986). The use of terms such as *anticipatory struggle*, *avoidance* or *accessory behaviors* imply intent and are unwarranted interpretations of speech behavior.

This distinction between two types of stuttering behaviors suggests that the stutterer has no voluntary control over the occurrence of sound/syllable repetitions and prolongations, but does have control over other types of behavior. It also implies that other abnormal behaviors, such as struggle, avoidance, and anticipatory behaviors are used by subjects to avoid or curtail the involuntary behaviors. The designation of some behaviors as core involuntary behaviors and others as "secondary" reactions to core behaviors could imply that one set of symptoms has a causal relationship to the other.

However, there is little to demonstrate that such a distinction can be made between sound/syllable repetitions and prolongations and other behaviors which have been interpreted as struggle, avoidance, or anticipatory behaviors. Few, if any, symptoms are common to all stutterers. Some adult stutterers rarely evidence sound/syllable prolongations and repetitions, but are severely communicatively impaired because of their frequent use of other behaviors often labelled anticipatory or avoidance behaviors. Further, sound/syllable repetitions and prolongations and anticipatory/avoidance behaviors occur in both normal children and adults when speech is momentarily interfered with by distraction, word-finding, etc. The difference between stutterers and normal speakers seems to be the frequency and duration of such speech disruptions. In children, a clearcut distinction between stuttering and normal behavior is lacking because many children exhibit the same behaviors as stuttering although less frequently (Conture, Chapter 1; Yairi, Chapter 6). Further, the frequencies and types of disfluencies change in stutterers over time making it difficult to determine which behaviors represent stuttering. Until we know more about the pathophysiology of stuttering, it seems premature to assign prominence of one type of symptom over another in the definition and measurement of the disorder.

If a broader definition of stuttering can be used for measuring the severity of impairment, stuttering will include all momentary disruptions in speech production when the speaker is not producing a target. This will include any disruptions, speech or other behaviors, which are not target speech. Such a definition makes no assumption regarding the cause or interpretation of the speech disruption. All behaviors which interfere with speech communication and disrupt the normal communication process would therefore be included in a measurement of severity. Such an approach would have content validity because it would measure the degree of disruption in communication. Stutterers whose only symptoms are frequent starters and a high percentage of retrials may be as communicatively impaired as those who have frequent syllable repetitions or sound prolongations. Both distract a listener, are time out from communication, and contribute to the listener's perception of problem severity.

To measure stuttering severity, therefore, all types of disruptions should be included by measuring the frequency and duration of disruptions and the duration of fluent intervals during communication. This is somewhat different from most of the methods currently being used to measure stuttering severity.

METHODS CURRENTLY BEING USED TO MEASURE STUTTERING SEVERITY

The types of instruments available for the measurement of stuttering severity include: examiner rating scales (the Lewis-Sherman scale as revised by Van Riper, 1982; the Riley Stuttering Severity Instrument, Riley, 1972); procedures for counting the frequency and duration of disfluencies (Costello & Ingham, 1984; Ham, 1986); and procedures for rating the naturalness of speech (Martin et al., 1984; Ingham et al., 1985). The research applicability of these instruments must be considered in terms of: the need for between and within subject comparisons; their suitability for use with children and adults; content and concurrent validity; ease of use; reproducibility between settings; and inter-and intra-examiner reliability.

Rating Scales

The Lewis and Sherman (1951) rating scales includes both sound/syllable repetitions and prolongations and postponement and avoidance behaviors. Three different speaking situations are included: talking for 3 minutes about job or vocation, reading a 300-word passage, and telling a story to a TAT card. The talking and story telling situations have the highest content validity. Sherman (1952) demonstrated that this scale had acceptable interrater reliability. Berry and Silverman (1972) and Van Riper (1982) made revisions in the interval scale and suggested it could be used for both between and within subject severity comparisons.

In contrast, the Riley Scale (Riley, 1972) which is more frequently used, has several limitations. First, whole word and phrase repetitions and rephrasing are not included in the estimate of the frequency of stuttering, limiting the content validity. Second, the rating scale requires judges to reliably discriminate in real time between "fleeting," one half and one full second disfluencies. Such discriminations may have poor inter- and intra-judge reliability. Third, frequencies of sound/syllable repetitions and prolongations are counted during both expository speech and oral reading in subjects above the third-grade level. Some milder subjects can stutter equally during conversation and oral reading, while others are severely impaired during conversational speech but not during oral reading. This instrument would equate the two. Because stuttering severity during reading is not related to severity during speech communication (Johnson et al., 1963), this detracts from the content validity of this instrument. Riley (1972) reported the interrater reliability was .84 although ratings of physical concomitants of stuttering such as eye blinks were less reliable (r = .62). Concurrent validity was demonstrated by a .89 rank correlation coefficient with the Iowa Scale of Stuttering Severity. The Riley scale can be used with young children as well as adults.

Measures of Frequency and Duration of Stuttering

Costello and Ingham (1984) present measures which can be made of stuttering during conversational/communicative speech, including: the percent of syllables stuttered, the average duration of stutterings, the duration of the 3 longest stutterings, overall speaking rate, articulatory rate for nonstuttered speech, the average duration of nonstuttered intervals, the duration of the 3 longest nonstuttered intervals, the length of the 3 longest nonstuttered intervals and naturalness ratings. The total number of syllables spoken within a time-period and the number of stuttered syllables are counted by button pressings while listening to the subject's expository speech. A counter-timer measures the length of time between button presses for stuttered speech. This provides a measure of the duration of noninterrupted speech. The length of 10 selected disfluencies are measured, as well as the 3 longest, with a stopwatch.

These measures of Costello and Ingham (1984) have good potential for providing objective measures of the frequency and duration of speech disruption. This approach has high content validity because communicative speech can be measured. However, no mention is made of the degree of inter- and intra-judge reliability of these measures. Studies of interjudge reliability in identifying disfluencies suggest that trained judges will only agree on 50% of the individual instances of disfluencies although their overall estimate of the total frequency of disfluencies will be more highly related (Curlee, 1981; Martin & Haroldson, 1981; Young, 1975). Further, the accuracy of these measures may also depend upon individuals' reaction times in starting and stopping the time-counter and stop watches. Some determination is needed of the percent error this can contribute to the measurement of disfluency duration, particularly for disruptions of less than a second.

The Stoker Probe method (Stoker, 1976) was developed for use with young children. It uses common everyday objects and examiner questions to elicit communicative speech. The examiner then tabulates the child's speech responses. This technique has high content validity because it attempts to sample the child's speech in response to varying degrees of communication pressure. Similar approaches, which should be standardized, are needed for older children and adults.

Naturalness Ratings

Naturalness ratings have received more attention in recent years, possibly because therapeutic approaches are more focused on changing the manner in which stutterers produce speech. Learning to change speech rate and produce "gentle" and "soft" voice onsets can have a marked effect on speech prosody. Successful graduates of these methods may not exhibit sound/syllable repetitions and prolongations but can be difficult to understand if their prosody is abnormal. Therefore, measures of severity, particularly before and after treatment, should include speech "naturalness" in addition to the frequency and duration of stuttering.

To study validity, Martin et al., (1984) used a 9-point rating scale and demonstrated that the scale could discriminate between normal speakers and stutterers with and without DAF. Interrater agreement was high with 75% of 13,050 pairwise comparisons of 30 judges' ratings either being identical or differing by only one rating unit. Similarly, repeated ratings were identical or differed by only one unit in 88%. This same rating scale was used reliably by Ingham et al. (1985), demonstrating reproducibility across settings. They found providing feedback of naturalness ratings to stutterers could be used to improve the naturalness of their speech.

NEEDS AND RECOMMENDATIONS FOR MEASURING STUTTERING SEVERITY

The lack of standardized, valid and reliable tools for assessing stuttering severity may be one of the roadblocks to research on this disorder. Such tools are necessary for quantifying the degree of disorder across subjects (between-subject measures) and for determining the effects of treatment (within-subject measures). When selecting assessment tools for research on this disorder, several considerations should be addressed.

A Single Continuum

Investigations into the pathophysiology of stuttering often relate measures of subjects' concurrent physiological attributes to the severity of their stuttering. For relating stuttering severity with other subject characteristics, between-subject severity measures are needed to rank subjects on one continuum. The interpretation of physiological differences would be strengthened if they related to standardized measures of the severity of subjects' speech disorders. Too frequently clinicians' subjective ratings are used which have no meaning to other investigators and can't be reproduced in other settings.

For evaluating treatment effects, the need for a single-continuum is less critical because comparisons are within-subject. However, a single continuous measure becomes necessary for determining if the degree of improvement is related to stuttering severity.

Reproducibility

Measures of stuttering severity are needed which can be used easily and have good interjudge reliability in different settings. That is, they must be reproducible by different investigators to compare their results. To build a knowledge base on the disorder, the same measures of stuttering severity should be used by different investigators. This is crucial, because stutterers vary. No two subjects exhibit the exact same speech problems and even the same individual varies in stuttering from moment to moment. Because physiological studies typically employ small numbers of subjects, usually less than 10, the results cannot be generalized to the stuttering population. Conflicting results may be the result of subject sampling. Use of reproducible measures by different investigators would allow comparison of the results across studies.

The use of the same assessment technique is also important in treatment studies. The amount of change in behavior with different treatments needs to be addressed. This will determine the relative benefit between approaches in well-defined subject subgroups.

Reliability

Because of the high variability in stuttering behavior within subjects, measures must have good stability over time when used repeatedly by the same examiners in the same setting.

Content Validity

Oral reading is not required for communication and the degree of speech impairment during oral reading is often not related to the degree of the subject's speech disorder in conversation (Johnson et al., 1963). Often the rationale for using oral readings is that it allows control of linguistic content and communication factors when controlled measures of treatment benefit are desirable. However, if oral reading is not a representative sample of a subject's speech impairment it may not have content validity for certain subjects. Such a controlled measure may not be an accurate picture of whether the communication disorder has been helped by treatment. Therefore, methods for measuring stuttering severity should be based primarily on communicative speech. For within-subject measurements, the speech task during which a subject stutters most frequently may have greater content validity. Because the degree of correspondence between impairment during oral reading and speech differs between subjects, measures during oral reading should not be used. This is particularly important when measuring treatment benefit because the most effective treatment should be one that improves a subject's communicative speech. Further, by restricting the measure to communicative speech, the same procedure can be used with both child and adult populations, regardless of reading development.

Some authors have argued for making measures in many different communicative situations to obtain representative measures of a subject's speaking behavior (Shames, 1976 as cited by Ham, 1986, p. 44; Ingham, 1981). In addition, consideration has been given to the need for covert measures of stuttering although these have not differed substantially from overt measures (Ingham, 1981). Stutterers have varying severity at different times in various situations. Therefore, to be truly representative of a stutterer's severity, multiple measures would need to be made in at least 6 different situations (e.g., face to face conversation with a stranger, with a close friend, telephone conversation, group situations, interviewing, and stress situations, Ingram, 1981). A more realistic solution might be to control the speaking situation by using a standardized communicative task for both inter- and intra-subject assessment.

A communication game task can be employed to acquire a standardized sample of communicative speech. Those requiring face to face communication with a listener with considerable communication demands might be effective. For example, the subject could be asked to direct a listener on how to construct a particular design. Such a task could be timed to add to the communication

stress. Different designs or similar items such as maps or block designs could be used which are appropriate to the subject's age, for use with children and adults. Similar tasks have been found useful in gathering speech samples in other communicatively impaired subjects (Ludlow, 1984). Research is needed to determine the concurrent validity of severity measures made during communication tasks with those made in other speaking situations.

Recording and Measurement

On-line ratings of naturalness should be most valid when the examiner is in the face to face situation. The examiner can determine on-line the degree to which speech interferes with communication by calling attention to itself or interfering with intelligibility. Similarly, the instances when communication is disrupted may be most easily identified by a listener in the face to face situation. However, the measurement of stuttering durations or type of disfluency needs to be done off-line from videotape. As discussed above, all disruptions in speech communication should be included when measuring the severity of stuttering.

For the speaker and listener, both the frequency and mean duration of fluent and nonfluent intervals may be important in determining the efficiency and ease of speech communication. Studies are needed to determine the inter- and intra-scorer reliability for the identification of disfluencies and other speech disruptions such as postponement and avoidance behavior and for estimating or measuring the duration of these behaviors. As Ingham (Chapter 11) suggests, the interval of uninterrupted communicative speech may be more reliably identified by scorers than individual events of speech disruption.

The measurement of fluent communicative speech can be performed using interval-timers or stop watches (Costello & Ingham, 1984). However, both inter- and intra-scorer reliability should be assessed for interval timing of fluent and disrupted speech both on-line and off-line from videotape. Further, such procedures may not be sensitive to changes in duration when speech disruptions are less than 1 second. To normalize across subjects with different rates of speech or who produce different size samples, the percentage of speech which is fluent or the percentage disrupted should be computed.

Improved methods of reliably identifying instances of speech disruptions should be developed. Studies of the effects of changing instructions to examiners to include the identification of any interruption in communication should also be explored.

Speech rate has been used for scoring stuttering severity. Because normal speakers differ considerably on this measure even when assessed during the same speaking task, this metric should not be used for intersubject assessment. Further, speech rate is often directly modified through treatment, so it may not be a valid intrasubject measure of changes in stuttering severity following treatment. For example, some subjects may produce fluent speech at 80 words per minute (wpm) while others may have a much lower percent of fluent speech but speak at 200 wpm. Some indication of these differences is needed and may be provided by naturalness ratings which are particularly important when assessing treatment outcomes.

Self-reporting of stuttering frequency has been found inaccurate (Ingham, 1981). However, covert stuttering may be the only symptom present in some subjects. To evaluate this, the use of a button press response by the subject whenever they judge a speech disruption to have occurred should be evaluated.

Scorer Training

For a measure to be useful for research, it must be easily reproducible across settings and not require extensive scorer training. Pretests for assessing scorer accuracy can assure that all scorers meet a minimal level of accuracy. Only those scorers meeting the criterion are then used to score subjects' videotapes. This might improve reproducibility across settings. When assessing treatment, scorers should be provided with coded videotapes randomly ordered to eliminate bias while scoring samples.

Repeated Sampling

For measurement methods to be useful for research they must be reliable, sensitive and valid. Because stuttering severity varies daily, several samples should be acquired (Ingham, 1981). Often a subject's speech is more fluent on the initial evaluation than on subsequent evaluations. Therefore, for comparing both within and between subjects, it is necessary to determine how many samples must be acquired to provide a stable measure of subjects' stuttering severity.

LIMITATIONS AND USES

The need for a single standardized measure of stuttering severity for between and within subject comparisons has been emphasized. Such an approach is recommended as a replacement for clinician severity ratings of "mild," "moderate," or "severe" when relating symptom severity to degree of physiological impairment. The reproducibility of clinician's ratings across research centers may depend, in part, upon the range in severity of subjects the clinicians have encountered. However, a single standardized measure of stuttering severity might not be as representative of a stutterer's overall communication impairment as a clinician's rating.

The communicative task has been suggested as a standardized speech situation for assessing treatment outcomes. Considerable effort will be required, however, to demonstrate its concurrent validity for representing a subject's degree of severity across many different speaking situations. However, the labor intensive and limited

reproducibility of sampling subjects in multiple settings has resulted in investigators using 3-point clinical rating scales for inter-subject comparisons. A standardized speech task might be the best compromise between the two approaches.

The lack of a standardized reproducible measure of stuttering severity has been a roadblock to comparing the degree of benefit with different treatment approaches. Further, treatment evaluations are often dependent upon data acquired during treatment sessions. The independence of the pre- and post-treatment measures from the treatment approach would allow for comparison of behavioral treatment approaches with no intervention, medical, drug, or placebo trials.

The types of measures discussed in this chapter are extremely crude. None provide information relevant to the identification or differential diagnosis of stuttering. The degree of impairment of a stutterer relative to normal speakers has not been addressed nor the differential diagnosis between stuttering, cluttering, and other types of communication disorders. Finally, no recommendations have been made regarding behaviors other than those which are overt and disrupt normal communication. A stutterer's circumlocutions to avoid certain utterances would only be noted if it interfered with normal speech communication.

However, at this stage in stuttering research, an objective standardized method of measuring severity in both children and adults who exhibit a variety of stuttering symptoms is necessary to increase our understanding and treatment of this disorder.

REFERENCES

BERRY, R., & SILVERMAN, F. (1972). Equality of intervals on the Lewis-Sherman Scale of Stuttering Severity. *Journal of Speech and Hearing Research, 15,* 185–188.

CONTURE, E. (1988). Childhood stuttering: What is it and who does it. *JFD,* in press.

COSTELLO, J., & INGHAM, R. (1984). Stuttering as an operant disorder. In R. Curlee & W. Perkins (Eds.), *Nature and treatment of stuttering.* San Diego: College-Hill Press.

CURLEE, R. (1981). Observer agreement on disfluency and stuttering. *Journal of Speech and Hearing Research, 24,* 595–600.

HAM, R. (1986). *Techniques of stuttering therapy.* Englewood Cliffs, NJ: Prentice-Hall.

INGHAM, R. (1981). Evaluation and maintenance in stuttering treatment: A search for ecstasy with nothing but agony. In E. Boberg (Ed.), *Maintenance of fluency.* New York: Elsevier North Holland.

INGHAM, R., MARTIN, R., HAROLDSON, S., ONSLOW, M., & LENEY, M. (1985). Modification of listener-judged naturalness in the speech of stutterers. *Journal of Speech and Hearing Research, 28,* 579–587.

JOHNSON, W., DARLEY, F., & SPRIESTERSBACH, D. (1963). *Diagnostic methods in speech pathology.* New York: Harper & Row.

LEWIS, D., & SHERMAN, D. (1951). Measuring the severity of stuttering. *Journal of Speech and Hearing Disorders, 16,* 320–326.

LUDLOW, C. (1984). Evaluation of treatment of children with language disorders. In W. Perkins (Ed.), *Current therapy of communicative disorders: Language handicaps in children.* New York: Thieme-Stratton.

ONSLOW, M., & INGHAM, R. J. (1987). Speech quality measurement and the management of stuttering. *Journal of Speech and Hearing Disorders, 52,* 2–17.

MARTIN, R., HAROLDSON, S., & TRIDEN, K. (1984). Stuttering and speech naturalness. *Journal of Speech and Hearing Disorders, 49,* 53–58.

MARTIN, R., & HAROLDSON, S. (1981). Stuttering identification: Standard definition and moment of stuttering. *Journal of Speech and Hearing Research, 49,* 59–63.

RILEY, G. (1972). A stuttering severity instrument for children and adults. *Journal of Speech and Hearing Disorders, 37,* 314–322.

SHERMAN, D. (1952). Clinical and experimental use of the Iowa Scale of Severity of Stuttering. *Journal of Speech and Hearing Disorders, 17,* 316–320.

STOKER, B. (1976). *Stoker Probe Technique for the Diagnosis and Treatment of Stuttering in Young Children.* Tulsa, OK: Modern Educational Corp.

STROMSTRA, C. (1986). *Elements of stuttering.* Oshtemo, MI: Atsmorts Publishing.

VAN RIPER, C. (1982). *The nature of stuttering.* Englewood Cliffs, NJ: Prentice-Hall.

WINGATE, M. (1964). A standard definition of stuttering. *Journal of Speech and Hearing Disorders, 29,* 484–489.

WORLD HEALTH ORGANIZATION, (1977). *Manual of the international statistical classification of diseases, injuries and caused of death* (Vol. 1). Geneva: World Health Organization.

YAIRI, E. (1988). Subtyping stuttering for research purposes: Children. *JFD,* in press.

YOUNG, M. (1975). Observer agreement for marking moments of stuttering. *Journal of Speech and Hearing Research, 18,* 530–540.

C. ETIOLOGY

Chapter 4

A REVIEW OF THE EVIDENCE FOR GENETIC FACTORS IN STUTTERING*

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CURRENT STATUS AND ROADBLOCKS

Stuttering is familial and there is evidence for vertical transmission in families (Kidd, Heimbuch, & Records, 1981). However, the mechanisms of that transmission are not clearly understood. A variety of hypotheses have been proposed, including several specific genetic models. Although there is evidence that genetic factors are important for the expression of stuttering, no specific type of genetic transmission has been elucidated.

A genetic factor for a disorder is demonstrated either by a specific structural or functional biochemical defect. No such evidence has been obtained for stuttering. In the absence of such data, there are at least four other types of studies which can provide support for genetic involvement in a disorder of unknown etiology: (a) twin studies; (b) family studies; (c) separation studies; and (d) genetic linkage studies.

Twin Study Method

The twin method consists of comparing the proportion of twin pairs in which both members are affected (i.e., the pair is concordant) in a sample of monozygotic (MZ) twins with the proportion of concordant twin pairs in a sample of dizygotic (DZ) twins. This method is used primarily to obtain preliminary evidence that genetic factors are important in the disorder being studied. The method can also be used to provide an estimate of the heritability of a particular disorder where heritability is defined as *the proportion of the total phenotypic variance of the trait being studied that is due to genetic factors.*

MZ twins are genetically identical. Thus, if differences between MZ twins occur, they can be due to genetic mutations or to different environmental influences experienced by the two developing individuals. Because genetic mutation of this type is an extremely rare event, the differences are usually assumed to be environmentally induced. DZ twins are no more closely related genetically than two siblings born at different times. Any differences between DZ twins are attributed to both genetic and environmental factors. Presumably, if DZ twins are more alike than two siblings, it could be due to shared environment. It should be emphasized that environmental influences begin at conception so that any environmental differences experienced prenatally could also influence the phenotype being studied. If it is assumed that same sex MZ and DZ twin pairs share equivalent environments, then any difference in concordance rates between MZ and DZ twin pairs are due to the fact that MZ twins are genetically identical whereas DZ twins are not.

Early twin studies of stuttering examined whether the prevalence of the disorder was increased among twins when compared to singletons. The hypothesis was that twinning in and of itself might be a risk factor for stuttering. No conclusive evidence was obtained. The rate of stuttering reported for twins varied considerably with Graf (1955) reporting a rate of 1.9% and Nelson, Hunter, & Walter (1945) reporting a rate of 20%.

Several other studies reported concordance rates for MZ and DZ twins. As is the case in many early twin studies for other disorders, there are concerns about the samples of twins obtained, the methods for zygosity determination, and the failure to control for sex (or simply report the sex of the twins). Thus, the results of the twin studies published must be interpreted with caution. For example, Nelson et al. (1945) report a 90% (9/10) concordance rate for MZ twins compared to a 6.6% (2/30) concordance rate for DZ twins. Although this difference is impressive, these authors did not provide information about the sex of the twins and hence it is not possible to determine if the difference may be inflated because of sex differences. For example, if the MZ twins were predominantly male and the DZ twins were not, then the differences could be exaggerated because females are less likely to stutter. Or if any opposite sex DZ pairs were included, with the male twin being affected, that pair

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would be more likely to be discordant because the co-twin is female and less likely to be affected in general.

In a study reported by Howie (1981), care was taken to control for some of these potential sources of bias. These investigators studied 30 same-sex twin pairs (21 male and 9 females). Seventeen of the twins were MZ and 13 DZ. Of the MZ twin pairs, 12 were male and 5 female. Among the DZ pairs, 9 were male and 4 female. The determination of zygosity was based on blood typing, finger print ridge counts, maximal palmar ATD angle, cephalic index and height. Concordance rates for stuttering were significantly higher for MZ twins (58%) compared to DZ twins (13%).

In another study conducted in an Italian population, Godai et al. (1976) reported concordance rates of 83% for MZ twins (N = 12 pairs) and 9% for same sex DZ twins (N = 11 pairs). Though both of these studies are quite small, together they yield evidence that genetic factors are important in the etiology of stuttering. If the two studies are combined (although it may not be appropriate to do so because of differences in diagnosis) concordance rates are 69% for MZ and 12% for DZ twins. Thus, because MZ concordance is significantly higher than DZ concordance, these twin study results are consistent with the hypothesis that genetic factors are important in the expression of stuttering. However, these results provide little information regarding the specific genetic mechanism(s) involved.

Family Study Method

Studies of biological families can also yield data suggesting genetic involvement for any given illness and can be used to test specific transmission hypotheses. The family study method consists of comparing rates of illness in families ascertained through an affected individual (the proband) with rates in the general population or with rates in families ascertained through unaffected persons (controls). If the risk of a disorder in families ascertained through an affected person is significantly greater than the risk of the disorder in the population or in the control families, the disorder is familial. This suggests a possible role of genetic factors for the illness. However, as with twin studies, if a major environmental component is important in the etiology of the trait in question, results drawn from family studies will be unable to prove the existence of genetic factors.

Data from families can, however, be used to demonstrate that vertical transmission occurs. Once vertical transmission has been established, the patterns of illness within families can be compared to those expected under a variety of specific genetic hypotheses. It is assumed that, if the pattern of the illness within families follows closely a pattern predicted by a classical Mendelian hypothesis, it is unlikely that environmental factors could be solely responsible for the transmission.

Van Riper (1971) summarized the results of a number of studies examining the families of stutterers. Most of these studies did not report the frequency of affected relatives. Instead they reported the frequency of positive family history. The median proportion of stutterers with a positive family history of stuttering was 42%, with a range of 24% to 80%. These kinds of data provide little evidence for genetic factors because the results are dependent on family size and ascertainment strategies. In addition, data on the frequency of positive family history cannot be used to test specific genetic hypotheses.

More relevant data are those which actually allow the determination of risk to relatives. Andrews and Harris (1964) reported an increased rate of stuttering among relatives of stutterers. In addition, they found that relatives of female stutterers were at greater risk than were relatives of male stutterers. Kay (1964) included information about sex of proband and sex of relative in the calculation of risks. Kidd and co-workers (Kidd, Kidd, & Records, 1978; Kidd et al., 1981) found similar risks among first-degree relatives of stutterers. In these studies, the overall risk for stuttering in first-degree relatives was about 15%. However, distinct differences were observed between the sexes. The overall rate of stuttering among relatives of females was 18% compared to about 14% among relatives of males. When the relatives were separated by sex, additional differences were observed. Stuttering occurred in approximately 20% of male relatives and 5% of female relatives of male stutterers. Among relatives of female stutterers, approximately 25% of male relatives and 12% of female relatives stuttered.

Thus, the available family data provide evidence that stuttering is familial and that specific patterns of vertical transmission occur which appear to be related to the sex of the proband and relative. Using this information, specific genetic hypotheses have been examined.

Because stuttering has long been observed to be familial, a number of genetic hypotheses have been proposed. Meyer (1945) and Andrews and Harris (1964) found several simple models of transmission, including autosomal dominant, recessive and X-linked inheritance, to be incompatible with the familial patterns observed. Kay (1964) suggested that either a single gene with polygenic background or a polygenic model might account for the data. Kidd (1977) showed that the patterns of transmission of stuttering in families were compatible with both a multifactorial-polygenic model and a single-major-locus model with sex-specific thresholds. Kidd's analyses incorporated only summary risk estimates for specific type of relatives. With these kinds of analyses, information regarding the specific pattern of transmission within each family is lost. Cox, Kramer, & Kidd (1984) suggested that discrimination among alternative genetic models might be possible with fuller utilization of the family data by segregation analyses.

Segregation analysis allows examination of the pattern of transmission in intact families and therefore has more power than previous methods which relied on summary frequency data. Cox et al. (1984) performed segregation analyses on a subset of the families studied by Kidd and co-workers (Kidd, 1977; Kidd et al., 1978; Kidd et al., 1981) and found that the transmission of stuttering observed in these families could not be adequately ex-

plained by segregation of a Mendelian major locus. However, the familial patterns could be explained by polygenic transmission.

These results need to be interpreted with care. Although the Yale Family Study of Stuttering is by far the largest to date, there are still a number of difficulties with the study. First, all of the data about first degree relatives were obtained through one informant. The vast majority of the first degree relatives were not seen and personally evaluated. In studies of other behavioral disorders, it has been shown that when rates of illness are calculated with data collected from just one informant they are consistently lower than the true rates of illness in families (Orvaschel, Thompson, Belanger, Prusoff, & Kidd, 1982; Pauls, Kruger, Leckman, Cohen, & Kidd, 1984). Moreover, this method of data collection can affect the pattern of illness observed within the families (Pauls et al., 1984). For example, it could be expected that an adult proband would be more likely to know about stuttering among his or her offspring than he or she would know about stuttering in parents or siblings. Thus, underreporting would occur for the parental generation as well as the sibling generation and result in different patterns within families than if all relatives were assessed and diagnosed.

A second shortcoming of these studies is that it is assumed in all of the genetic analyses that the trait being studied is etiologically and genetically homogeneous. Given what is known about stuttering, this assumption is most likely wrong.

Taken together these two difficulties weaken the conclusions of this study. For example, Cox et al. (1984) indicate that in 54% of the families, the index case was the only affected individual in the family and suggest that the relatively high prevalence of stuttering in the population and the high proportion of isolated cases in these families may effectively prevent these analyses from yielding evidence for the presence of a major gene involved in stuttering. Given that not all first-degree relatives were personally assessed it is not possible to know whether the isolated cases were truly singletons or whether other relatives were missed because of inadequate assessment. If some of the probands included as isolated cases actually had affected relatives, then the results of the segregation analyses would most likely differ from those reported by Cox et al. (1984). Thus, we do not have convincing evidence to rule out any specific genetic hypothesis.

Even more detrimental is the assumption of homogeneity of stuttering. Given what is known about other genetic illnesses, a disorder as common as stuttering is almost certain to be etiologically and genetically heterogeneous. If stuttering is heterogeneous then the assumption of homogeneity would invalidate all of the segregation analyses performed.

Other Methods

As indicated earlier, there are at least two other methods available which provide evidence for genetic factors: (a) separation studies and (b) linkage studies. Neither type of study has been applied specifically to stuttering. Although both can be quite useful approaches, the genetic linkage methodology in particular deserves additional discussion.

Genetic linkage is detectable, at least in theory, if a known genetic marker locus is sufficiently close to a locus affecting the trait under study so that non-random segregation of alleles at the two loci results in an association of phenotypes within a family. The demonstration of genetic linkage requires family studies showing that alleles at two separate loci are physically close on the same chromosome. Family data are used to estimate how frequently the alleles at the two loci are transmitted to a child in combinations different from those in the parents. The degree of linkage is measured as the recombination fraction (the frequency of such new combinations) and can range from zero (complete linkage) to 0.5 (independent assortment). The minimum recombination frequency of zero is found for alleles that are always transmitted in the same combinations from generation to generation. The maximum recombination frequency of 0.5 is found for alleles (at two separate loci) that have the same likelihood of being transmitted in new combinations as in the same combination from generation to generation. This maximum recombination frequency occurs for alleles at loci far apart on the same chromosome and, of course, for alleles at loci on different chromosomes. Hence, maximum recombination is just another way of phrasing Mendel's second law of independent assortment. Linkage results in the violation of that law.

Some methodological problems in detecting linkage in human data include small family sizes, the inability to control matings, and the small prior probability that the two loci are linked. Historically, the method has had limited applicability chiefly because of the small number of sufficiently polymorphic genetic markers that were available for humans. This has changed rapidly because of the advance in genetics brought about by recombinant DNA techniques. A new class of polymorphisms has been identified. This class of polymorphisms is referred to as "restriction fragment length polymorphisms" (RFLPs) because they are visualized as inherited variations in the length of defined fragments of DNA when it is digested with specific restriction enzymes. For an excellent review of the methods involved in identifying these polymorphisms and their potential use for the genetic study of stuttering see Cox (1988).

Using these new polymorphisms as markers, investigators are nearing completion of a genetic map of the entire human genome (Helms, Green, Weiffenbach, Bowden, Keith, Stephens, Smith, Akots, Bricker, Brown, Gravius, Morgan, Muller-Kahle, Phipps, Rising, Ng, Rediker, Powers, Falls, Hogen, Cannizzaro, & Donis-Keller, 1988). Markers mapped in this way have been extremely useful in mapping other disease loci (e.g., Huntington's disease (Gusella, Wexler, Conneally, Naylor, Anderson, Tanzi, Watkins, Ottina, Wallace, Sakaguchi, Young, Shoulson, Bonilla, & Martin, 1983) and Alzheimer's disease (St. George-Hyslop, Tanzi, Polinsky, Haines, Watkins, My-

ers, Feldman, Pollen, Drachman, Growdin, Bruni, Foncin, Salmon, Frommett, Amaducci, Sorbi, Piacentini, Stewart, Hobbs, Conneally, & Gusella, 1987). It should be anticipated that this methodology will also be useful in attempts to learn more about the underlying genetic factors which may be important for the expression of stuttering.

IMPLICATIONS FOR FUTURE RESEARCH

As is evident from this brief review, little is known about the genetics of stuttering. New family studies are needed which use state of the art methods. The most important area to be addressed is the assessment of phenotype. In addition to assessing carefully the proband, all members of the families need to be evaluated personally. This evaluation must be able to identify all individuals who have ever stuttered. Because so many stutterers become fluent speakers later in life, valid and reliable means have to be developed to diagnose stuttering using retrospective information. It is critical in a family study to know every person who has stuttered at some period in their lives. Only with data like these will it be possible to test with confidence specific genetic hypotheses.

Additional studies are also needed which attempt to identify possible subtypes of stuttering. If there are etiologically distinct subtypes of stuttering, it is critical to the success of any family study that those subtypes be identified so that families may be separated into clinically homogeneous groups. In fact, if the study is large enough, family data may be useful in identifying unique subtypes. For example, suppose that 50% of all cases of stuttering are isolated cases (i.e., they have no family history of stuttering). By examining those individuals and comparing them to familial cases, some clinical differences might emerge. Further division (e.g., by a pattern of transmission within families) might yield additional characteristics useful in separating stutterers further into distinct clinical subtypes. Unique subtypes would enhance the chances of understanding more fully the etiology of this disorder and would ultimately lead to better approaches to treatment.

Only after studies of the type just discussed have been undertaken will it be possible to fully utilize the power of genetic linkage studies. Accurate phenotypic assignment is absolutely essential to the success of a genetic linkage study. Again, valid and reliable methods of assigning a lifetime diagnosis of stuttering are critical.

Linkage studies require large multigenerational families with several affected individuals. Because it is quite likely that stuttering is genetically heterogeneous, the families should be large enough to achieve statistical significance with data from just one family. However, several large families should be collected so that replication is possible.

Finally, because environmental factors undoubtedly play a role in the expression of stuttering, prospective studies of children at risk should be initiated. By following children over time it should be possible to identify potential risk factor associated with the onset and expression of the disorder. Attempts should be made to collect information regarding all possible factors thought to be associated with the expression of stuttering. These studies should be initiated as soon as possible in families where there is a family history of stuttering. In families of this kind, there is a greater chance that genetic factors are important. It will then be possible sometime in the future, if a genetic locus is identified via linkage studies, to actually identify all people in these families who are at risk genetically and to identify any possible non-genetic factors important for the ultimate expression of the disorder. By combining these two approaches the chances of identifying critical risk factors will be increased.

REFERENCES

ANDREWS, G. E., & HARRIS, M. M. (1964). *The syndrome of stuttering* (Clinics in Developmental Medicine, No. 17). London, Spastics Society Medical Education and Information Unit in Association with William Heinemann Medical Books.

COX, N. J. (1988). Molecular genetics: The key to the puzzle of stuttering? *Journal of the American Speech-Language-Hearing Association, 30,* 36–40.

COX, N. J., KRAMER, P. L., & KIDD, K. K. (1984). Segregation analyses of stuttering. *Genetic Epidemiology, 1,* 245–253.

GRAF, O. I. (1955). Incidence of stuttering in twins. In W. Johnson (Ed.), *Stuttering in children and adults.* Minneapolis: University of Minnesota Press.

GODAI, U., TATARELLI, R., & BONNANNI, G. (1976). Stuttering and tics in twins. *Acta Genetica Medicae et Gomellologiae, 25,* 369–375.

GUSELLA, J. F., WEXLER, N. S., CONNEALLY, P. M., NAYLOR, S. L., ANDERSON, M. A., TANZI, R. E., WATKINS, P. C., OTTINA, K., WALLACE, M. R., SAKAGUCHI, A. Y., YOUNG, A. B., SHOULSON, I., BONILLA, E. & MARTIN, J. B. (1983). A polymorphic DNA marker genetically linked to Huntington's disease. *Nature, 306,* 234–238.

HELMS, C., GREEN, P., WEIFFENBACH, B., BOWDEN, D., KEITH, T., STEPHENS, K., SMITH, D., AKOTS, G., BRICKER, A., BROWN, V., GRAVIUS, T., MORGAN, D., MULLER-KAHLE, H., PHIPPS, P., RISING, M., REDIKER, K., POWERS, J., FALLS, K., HOGAN, C., CANNIZZARO, L., & DONIS-KELLER, H. (1988). A human genetic map with 545 genetic loci. *American Journal of Human Genetics, 43,* (Supplement), A147.

HOWIE, P. M. (1981). A twin investigation of the etiology of stuttering. *Journal of Speech and Hearing Research, 24,* 317–321.

KAY, D. W. K. (1964). The genetics of stuttering. In G. E. Andrews, & M. M. Harris (Eds.), *The syndrome of stuttering.* London: William Heinemann Medical Books.

KIDD, K. K. (1977). A genetic perspective on stuttering. *Journal of Fluency Disorders, 2,* 259–269.

KIDD, K. K., HEIMBUCH, R. C., & RECORDS, M. A. (1981). Vertical transmission of susceptibility to stuttering with sex-modified threshold. *Proceedings of the National Academy of Sciences, 78,* 606–610.

KIDD, K. K., KIDD, J. R., & RECORDS, M. A. (1978). The possible causes of the sex ratio in stuttering and its implications. *Journal of Fluency Disorders, 3,* 13–24.

MEYER, B. C. (1945). Psychosomatic aspects of stuttering. *Journal of Nervous and Mental Disorders, 101,* 127–157.

NELSON, S. E., HUNTER, N., & WALKER, M. (1945). Stuttering in

twin types. *Journal of Speech Disorders, 10,* 335–343.

ORVASCHEL, H., THOMPSON, W. D., BELANGER, A., PRUSOFF, B. A. & KIDD, K. K. (1982). Comparison of the family history method to direct interview: Factors affecting the diagnosis of depression. *Journal of Affective Disorders, 4,* 49–59.

PAULS, D. L., KRUGER, S. D., LECKMAN, J. F., COHEN, D. J., & KIDD, K. K. (1984). The risk of Tourette's syndrome and chronic multiple tics among relatives of Tourette's syndrome patients obtained by direct interview. *Journal of the American Academy of Child Psychiatry, 23,* 134–137.

ST. GEORGE-HYSLOP, P. H., TANZI, R. E., POLINSKY, R. J., HAINES, J. L., WATKINS, P. C., MYERS, R. H., FELDMAN, R. G., POLLEN, D., DRACHMAN, D., GROWDIN, J., BRUNI, A., FONCIN, J. F., SALMON, D., FROMMETT, P., AMADUCCI, L., SORBI, S., PIACENTINI, S., STEWART, G. D., HOBBS, W. J., CONNEALLY, P. M., & GUSELLA, J. F. (1987). The genetic defect causing familial Alzheimer's disease maps on chromosome 21. *Science,* 235, 855–890.

VAN RIPER, C. (1971). *The nature of stuttering.* Englewood Cliffs, NJ: Prentice-Hall.

Chapter 5

FACTORS IN THE ETIOLOGY OF STUTTERING

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If we have one clear fact to aid us in the attempt to understand the role of factors other than genes in the etiology of stuttering, it is this: pairs of identical twins, for whom zygosity has been carefully determined, are not always concordant for stuttering (Howie, 1981). Although the evidence from the study of twins clearly supports a genetic hypothesis, Howie's finding that 6 of 16 monozygotic twin pairs were discordant for stuttering demonstrates that genetic factors alone are not sufficient to produce stuttering. Some processes that occur after the specification of genetic material must be critical to the development of stuttering. The goals of this paper are to review current knowledge of the role of these etiological factors, and, on the basis of this review, to indicate needs and directions of future research.

A ROADBLOCK: THE LACK OF A COMPREHENSIVE THEORETICAL FRAMEWORK

An obvious place to look for summaries of factors significant for the development of stuttering is in theoretical accounts of stuttering. Theories, by definition, should summarize the available data into a coherent framework. There have been some attempts to develop integrated theories of stuttering (e.g., Starkweather, 1987b, Zimmermann, 1980; Zimmermann, Smith, & Hanley, 1981, and this conference), however, there is not a generally accepted theoretical framework that unites experimental results into a working model of stuttering. This presents a major roadblock to progress in understanding stuttering. Although it is clear that we cannot expect, nor would we want, full agreement among the community of scientists interested in this problem, we might expect that there would be a common set of goals for stuttering theory. Theories might differ in the conditions thought to be related to certain phenomena, but it would seem that the various theories ought, at least, to be attempting to explain a common set of phenomena associated with stuttering. In other words, as Zimmermann and Kelso (1982) have argued, we should be able to agree on what it is we would want to *explain* with a theory of stuttering.

The lack of a cohesive framework for stuttering research seems to be due in large part to a neglect of metatheoretical issues. Two major problems exist: (a) the pervasiveness of misguided concepts of causality and (b) isolation of stuttering research and theory. Almost 20 years ago, Perkins and Curlee (1969) outlined and discussed numerous flaws in ideas of causality that were rampant in the field of speech pathology. They concluded, "muddled thinking about cause and effect doubtlessly helps preserve muddled conceptions of the problems for which we hold professional responsibility" (p. 238). More recently, this issue has been taken up specifically in relation to stuttering research and theory by Zimmermann and Kelso (1982). Following Woodger (1952), Zimmermann and Kelso suggest that words or phrases such as "determine," "due to," or "cause of" are best omitted from discussions of stuttering. They state: "The issue is not if genes or environments cause stuttering. The only testable issues are what environmental conditions and what biological conditions are associated with the development of stuttering" (p. 4).

A second metatheoretical problem is the frequent isolation of stuttering research and theory. MacKay and MacDonald (1984) also have been critical of the metatheory underlying past stuttering research. They state, "Under this metatheory, stuttering can be studied by itself, independent of both data and theories on how normal, error-free speech is achieved. In short, this metatheory views stuttering research as a field unto itself with its own special methodology, phenomena, and theories" (p. 262). In addition to being isolated from the study of other behaviors, stuttering theories have been isolated from each other. A variable held to be of critical importance to stuttering by one theorist may not even be mentioned when another theorist constructs his or her model.

Although these metatheoretical problems have been described and discussed, they persist. It is likely that their persistence reflects the lack of a theoretical framework that can be generally agreed upon as a preliminary

basis for the investigation of stuttering. Zimmermann and his colleagues (Zimmermann, 1980; Zimmermann, Smith, & Hanley, 1981) have proposed a unifying theoretical framework for research and theory in stuttering. Their view is that there are many types of variables that will ultimately be of interest in the attempt to understand stuttering: sociocultural, psychological, genetic, physiological, and other variables we may not be able even to guess at this time. Despite the number and complexity of the variables likely to be on this list, a unifying framework is provided by the realization that, in order to play a role in stuttering, each of these variables ultimately must directly or indirectly influence speech motor control processes. Therefore, if the claim is made that expectation or arousal plays a significant role in stuttering, it is essential to demonstrate how these factors could interface with the neural mechanisms responsible for the control and coordination of speech movements. Such assertions derive from the fact that stuttering involves a breakdown in motor control processes. To Zimmermann and colleagues (1981), this is a central and unifying aspect of the disorder.

The publication of papers from a recent conference under the title, *Speech Motor Dynamics in Stuttering* (Peters & Hulstijn, 1987), suggests that investigators of stuttering are more and more interested in the potential significance of physiological measures and their interpretation within a motor control perspective. Yet many writers appear to view the unifying framework of Zimmermann et al. (1981) as a statement that the "cause" of stuttering is to be found in the motor system. For example, Andrews, Craig, Feyer, Hoddinott, Howie, and Neilson wrote in their 1983 review, "In contrast to Zimmermann, who sees stuttering as a purely motor disorder, our present theoretical position, now to be outlined, focuses on the central processing subserving speech production" (p. 238). Another example suggests that linguistic factors are left out of "motoric views" of stuttering; "While our view is that the weight of evidence favors the interpretation that stuttering is characterized by intermittent language-speech formulation irregularities, future research may support Zimmermann's hypothesis that impaired motor (kinematic) feedback accounts best for stuttering phenomenology" (Hamre, 1984, p. 250–25). Such comments are made despite the fact that Zimmermann (1980) explicitly included in his model of stuttering the following components: linguistic constructs, cognitive representation, auditory and spatial coding, descending influences from cortical and cerebellar sites, learning, adaptation, as well as reflex gains and articulator velocity or displacement. What has happened is that the level of measurement used, peripheral measures such as kinematics and EMG, is misinterpreted as the level of explanation sought. Many people believe that the analysis of peripheral physiological events will be useful to the attempt to understand stuttering, but clearly this is only one level of description that will be needed to clarify the conditions that converge to produce stuttering.

My purpose in discussing these issues is not to encourage acceptance of Zimmermann's model. Indeed we are so far from being able to specify many of the relationships outlined by Zimmermann and colleagues, (e.g., the relationship between emotional factors and coordinative breakdown), that the model is perhaps better viewed as a conceptual framework. Clearly there are competing views on what would constitute an appropriate framework for the investigation of stuttering. In a very recent paper, "Stuttering, Delayed Auditory Feedback, and Linguistic Rhythm," Harrington (1988) proposed a model of stuttering in which the role of auditory information in generating rhythmic structures of speech is the central component. He argues that "the main advantage of this kind of model is that it provides a convenient metalanguage in which several diverse theoretical and experimental findings can be related" (p. 44). I would argue that, while the role played by auditory information in generating normal and disfluent speech is an important issue, this "model" of stuttering is far too narrow to provide a framework for discussing all of the factors that play a role in stuttering. If we cannot agree on the best framework for research in stuttering, we could at least begin to generate a set of goals for stuttering theory, to specify what it is we think should be explained by a theory or model of stuttering (Zimmermann & Kelso, 1982).

The preceding discussion of theoretical roadblocks may seem to be a digression from etiological factors in stuttering. Actually, it is precisely the issue of etiology that brings these theoretical problems to the surface. A review of ideas and experimental results that address the etiology of stuttering makes it is clear that inappropriate searches for "causes" of stuttering have produced a confused and confusing literature. There have been repeated attempts to isolate the "core" behaviors of stuttering from "secondary" or "associated" behaviors. The logic seems to be that the "core" behaviors are truly stuttering, while the "associated" behaviors develop as a "consequence" or "reaction to" stuttering. Examples of such thinking are numerous. In reviewing personality factors in stuttering, Andrews et al. (1983) wrote, "Stutterers do show more difficulties than nonstutterers with social adjustment (A) (Brown & Hull, 1942; Prins, 1972; Wingate, 1962), but this finding is probably a consequence rather than a cause of stuttering" (p. 230). Many writers have debated whether deficits in laryngeal control are a "core" behavior of stuttering. Adams (1984) reviewed studies of voice initiation and termination and found evidence of slowness both in adult and child stutterers. He concluded that these findings would not be likely if "stuttering were the cause of the slowness. Rather, such slowness probably antedated or coincided with the onset of the disorder. Indeed, it is even possible that difficulty in quickly initiating voicing is one of the immediate causes of stutterers' repetitions and prolongations of articulatory gestures (Adams, 1974), viewed here and elsewhere as core characteristics of stuttering" (Wingate, 1964) (p. 101).

Carrying this debate one step further, Stromsta (1987) weighed experimental evidence to decide whether "phonatory disturbance is a correlate of stuttering or a conse-

quential reaction to stuttering" (p. 267). He argued that it is not phonatory disturbance but "intraphonemic disruption" that is the "core behavior of stuttering—or primary stuttering—due to faulty coarticulation" (p. 267). Following this line of logic, Stromsta's final conclusion is this:

> Let us assume for the moment something we believe to be true, that advanced stage stutterers characteristically minimize their innate core behavior repetitions by adopting a host of variable reactions including prolongations and tonic blocks. If this is true, then the continued unqualified use of advanced stage stutterers as research subjects in attempting to describe and define the basic nature of the disorder will continue to 'muddy the waters' of theory, research, and therapy of stuttering. Because of this, at least in our opinion, the literature of stuttering, while being a commendable source of information related to reactions to stuttering, verges on being vacuous concerning the acoustic and the neuromuscular output signals of the core behavior of stuttering. (p. 217)

Do problems with social adjustment cause stuttering or does stuttering cause problems with social adjustment? Does a deficit in laryngeal functioning cause stuttering or does stuttering cause a deficit in laryngeal functioning? Does faulty production at the laryngeal level cause aberrant articulatory events or does aberrant articulatory control cause problems with control of laryngeal events? Or perhaps both laryngeal and articulatory problems in stuttering are caused by respiratory malfunction. Does a generalized deficit in language skills cause stuttering or does stuttering cause a generalized deficit in language skills? One could generate an unending list. Such questions, however, do not lead to testable hypotheses. The inevitable futility of pursuing such questions is perfectly illustrated by Stromsta's conclusions that prolongations are caused by stuttering and that studies of adult stutterers cannot provide any information about the nature of the disorder. In general we must look for conditions associated with stuttering and not attempt to attribute causal status to one variable or another.

The idea of the "core" behaviors of stuttering seems to have contributed to misguided concepts of causality and perhaps should be abandoned. Stuttering is a progressive disorder in the sense that it changes over time (Bloodstein, 1981). The claim that the "core" behaviors of stuttering are those that emerge first and that cause subsequent behaviors seems analogous to reasoning that, if the first symptom of a cold is a sneeze, then sneezing is the "core" behavior of colds, and that symptoms arising later are caused by sneezing. The entire complex of behaviors associated with stuttering in children and in adults *is* stuttering. There may be conditions associated with stuttering at an early age that later tend to be absent, or there may be aspects of stuttering in adults that are generally not present in children. In addition, there may be some conditions associated with stuttering that are more common than others, but these should be seen simply as more frequent, more reliable indicators of the disorder, rather than as causal.

Having repeatedly alluded to "the conditions associated with stuttering," I now am bound to turn to the question of what these conditions are. Pauls (Chapter 4) has outlined the evidence for genetic factors in stuttering. At first, it might seem that my task would be to consider "nongenetic" factors. This is not the case, because many of the factors to be discussed, for example personality or emotions, clearly have a genetic base. As Pauls' review indicates, we have no idea what genes are involved in the transmission of a predisposition to stuttering. Recognizing this fact, the following review includes factors not directly discussed in the "genetic" section and rests on the assumption that each of these factors evolves from the complex interaction of genes and environment.

CURRENT STATUS: A REVIEW OF EXPERIMENTAL RESULTS

Many types of factors have been proposed as significant for the development of stuttering. These have been reviewed in detail by Bloodstein (1981), Andrews et al. (1983), and Van Riper (1982). They include: emotional states such as anxiety and fear; communicative stress and parental and other listeners' attitudes and perceptions; learning in various forms, for example, acquiring beliefs about communicative skills or classical conditioning of negative emotion in response to speech-related stimuli; genetically transmitted or acquired deficit in sensorimotor skills; language and medical histories; and cultural factors. Because the literature relating to these factors is vast and has been reviewed previously, the present focus will be on studies completed in the last 5 years and their relationship to broad, historical perspectives on the etiology of stuttering.

Emotional States

Emotion has played an important role in many theories of stuttering (Brutten & Shoemaker, 1967; Johnson, 1955; Sheehan, 1970; Wischner, 1952). In general it is suggested that anticipation of speech difficulty produces a negative emotional response, such as fear or anxiety. Zimmermann (1980) has suggested that emotional state or arousal may influence somatic sensorimotor functions via autonomic pathways. There is also the speculation that factors such as expectancy, fear, and anxiety are not present in the very young stutterer, but that their influence on speech behavior of stutterers increases with age (Bloodstein, 1981; Van Riper, 1982).

One way the potential relevance of psychological state variables to stuttering has been tested is by comparing stutterers' and normal speakers' personality attributes, particularly and anxiousness and neuroticism. Reviewers of studies comparing both children and adults have generally agreed that there are no differences in personality factors of stutterers vs. nonstutterers (Andrews et al., 1983; Van Riper, 1982). However, in a recent study by Peters and Hulstijn (1984), there were significant differences between stutterers and nonstutterers in both failure

anxiety scales of the Prestatie Motivatie Test. In addition, these investigators found that stutterers' ratings of their subjective anxiety after performance of speech tasks were significantly higher than those of nonstutterers. These results may suggest that more sophisticated personality tests, in which specific aspects of anxiety are tapped, may reveal consistent differences between stutterers and nonstutterers.

Despite the generally negative findings in the search for the "stuttering personality," the belief persists that emotion or arousal is a significant factor in stuttering. Although empirical support for this idea is lacking, self-reports of stutterers and the weight of clinical evidence suggest that emotional state variables are important (Van Riper, 1982). A problem with self-reporting and personality testing is that they require one to invoke constructs such as "anxiety." The well-known difficulty in defining "anxiety" still plagues psychologists.

Another approach that may clarify the role of emotional factors and that avoids problems of definition is to measure physiological variables, for example, skin conductance, pulse volume, and heart rate, that are related to autonomic functions (e.g., Martin & Venables, 1980). This seems reasonable, because it seems likely that, if psychological state variables play a role in producing the coordinative breakdowns characteristic of stuttering, they do so via changes in autonomic function (e.g., Zimmermann, 1980). Measurement of indices of autonomic function in stutterers has produced mixed results, but with enough convincing positive findings to make this a potentially valuable area to pursue. Evidence of autonomic arousal associated with stuttering has been suggested by decreases in peripheral blood volume (e.g., Ickes & Pierce, 1973) and large increases in levels of urinary catecholamines following stressful speaking situations (Chmelova, Kujalova, Sedlackova, & Zelany, 1975; Edgren, Leanderson, & Levi, 1970). Baumgartner and Brutten (1983) found that mean heart rate measured prior to speaking a word was correlated with the number of disfluencies in only one of three subjects tested.

Perhaps the most complete study of autonomic measures in relation to stuttering has been reported by Peters and Hulstijn (1984). They simultaneously measured heart rate, skin conductance responses, and digital pulse volume of 24 adult stutterers and 24 nonstutterers before, during, and after performance of a number of different tasks. The tasks included writing, mirror writing, performance of items from an intelligence test, silent reading, reading, and conversational speech. The major result of this study was that in both groups, stutterers and nonstutterers, more physiological responses were observed before and during speech tasks compared to the other tasks. Within the speech tasks physiologic indices of arousal were highest for conversational speech. Analyses of variance did not produce any convincing group differences, and Peters and Hulstijn concluded there were "only minor differences" between the two groups and noted a high degree of individual variability. Their results, however, are important in that they suggest that increased autonomic arousal is associated with speech regardless of whether the speaker is a nonstutterer or stutterer. Increased autonomic arousal considered in isolation from other variables may not be a sign of abnormal functioning, but in the stutterer this increased arousal in conjunction with other factors may be contributing to breakdown.

In summary, studies of adult stutterers provide no clear picture on the question of whether autonomic nervous system activity is associated with stuttering. In view of its theoretical significance, this is an important issue to resolve. Much of the early work in this area is difficult to interpret due to nonstandardized measurement techniques. Now standardized methods of measurement of these psychophysiological variables have been outlined in detail (Martin & Venables, 1980) and should be used in the investigation of stuttering. In addition to using standardized techniques of measurement, such studies should avoid looking only at differences between groups of stutterers and nonstutterers. Previous results (Baumgartner & Brutten, 1983; Peters & Hulstijn, 1984) suggest that the strength of the relationship between indicators of autonomic activity and stuttering may vary between individuals.

The relationship between emotion or autonomic activity and stuttering at earlier ages is unknown, but the idea that such variables are not associated with stuttering in young children has been expressed by a number of writers (e.g., Bloodstein, 1981). This idea seems to be consistent with Johnson's (1942) notion that the disfluencies of early childhood are more "normal" than those of the adult, and that emotional reactions to disfluency and anticipation of speech difficulties arise later. Experimental support for this hypothesis is weak. In summarizing data gathered from a large number of stutterers between the ages of 2 and 16, Bloodstein (1960a,b) provided evidence that features of stuttering such as word or sound fears, anticipation, and avoidance of speech did not appear until after age 8. Van Riper (1982) disagreed with this picture of a uniform, gradual onset without emotional or physical struggle and proposed that there were different types or "tracks" of development in stuttering. More recent evidence provided by Yairi (1983) supports Van Riper's position. Yairi's data indicated that in very young stutterers, 22 children aged 2–3 years, 4 of the children were "aware and bothered" by their stuttering when it first began. Further, 15 of the children used "force or more effort" to speak when stuttering began. Thus in children as in adults, the relative importance of emotional factors may vary between individuals.

Communicative Stress, Including Parental and Other Listeners' Attitudes and Perceptions

A large number of variables has been measured to address the role of such factors in stuttering. For example, this category would include "linguistic" variables, such as sentence length, syntactic complexity, lexical control (e.g., Hegde, 1982), and effects of listener variables, such as number of interruptions, and audience size and com-

position. Particularly in the realm of linguistic variables, a number of recent investigations have added to the growing evidence (Van Riper, 1982) that these factors are significant in stuttering behavior. For example, Jayaram (1984) demonstrated effects of sentence length and clause position on the occurrence of disfluencies of adult stutterers. Bernstein-Ratner and Sih (1987) used an elicited imitation task to evaluate effects of utterance length and syntactic complexity in normal and stuttering children, aged 3 to 6 years. Fluency breakdown was strongly correlated with syntactic complexity for both groups of children, while utterance length showed much weaker correlations with measures of fluency.

With numerous studies producing similar results, the present challenge is to determine how these variables affect speech motor control processes. An obvious hypotheses is that linguistic "loading" coincides in time with increased demands on motor programming. In view of experimental results suggesting that stuttering is associated with a deficit in the ability to program and initiate movements (e.g., Peters & Hulstijn, in press), this is an attractive hypothesis. Another possibility, derived from the idea that stutterers have a limited capacity for certain "sensorimotor information processing" (Neilson & Neilson, 1987), is that fluency is likely to be compromised when any additional demands on central processing are made. Thus, demands for linguistic processing have no special status. Any requirement for processing concurrent to the production of speech will increase the likelihood of breakdown. As Neilson and Neilson (1987) suggest, this hypothesis leads to potentially rewarding experiments using various dual task paradigms. These are issues that should be taken up in studies of both children and adults who stutter.

Another form of communicative stress thought to be significant in disfluent behavior may be generated by the responses of the listener. Of special importance to the development of stuttering are the attitudes and responses of parents. Johnson's (1942) influential theory placed the "blame" for the onset of stuttering squarely on the shoulders of the parents. Clearly for both ethical and therapeutic reasons this is an important issue. There are no clear-cut empirical results that support the contention that certain familial attitudes or patterns of verbal behavior are associated with an increased risk for the development of stuttering. Writing on treatment of young chronic stutterers, Costello (1984) concluded, "No rigorous experimental evidence exists to demonstrate that the presence or absence of any family-environment variable or composite of variables is a functional antecedent to stuttering" (p. 377). In fact, some experimental evidence has indicated that parents of stutterers do not behave in ways that have been assumed to be detrimental for the development of speech in their children. A recent study by Meyers and Freeman (1985a) found evidence that mothers of nonstuttering children interrupted the disfluent speech of stutterers significantly more often than did mothers of stutterers. Clearly there is a need for additional quantitative assessment of the association of certain parental behaviors and the emergence of stuttering.

The role of listeners other than parents also has been examined in relation to stuttering. Again, the data do not provide clear support for the idea that the behavior or attitude of the listener has any consistent relationship to the behavior of the stutterer. In 2-, 4-, and 6-year-old nonstuttering children, Wexler (1982) compared neutral and stressful (repeated interruptions by the listener) speaking conditions. For the 4- and 6-year-old groups no differences were found, while in the 2-year-old children, the number of disfluencies was lower in the stressful compared to the neutral condition. Perhaps the most convincing evidence that listeners may have an effect on the speech of adult stutterers comes from a study by Martin and Haroldson (1988). Percent of words stuttered increased significantly when the stutterers conversed with a listener present compared to scores derived from spontaneous speech produced without a listener.

Learning

For many years the debate raged whether stuttering was a "learned" or an "organic" disorder. Happily this debate has passed, and at present I do not think anyone would argue with the statement that learning plays a role in the development of stuttering. However, learning theories as a basis for understanding stuttering (e.g., Brutten & Shoemaker, 1967; Sheehan, 1953) have been largely abandoned, because the level of explanation that ultimately will be required for understanding this disorder cannot be achieved within a purely behavioral framework (Zimmermann & Kelso, 1982). In other words, to state that stuttering arises as an approach-avoidance conflict or that stuttering is an operant behavior does not adequately explain stuttering. "Learning" does not really designate a separate class of variables in this list of putative etiological factors, because learning is embedded in every factor. In any discussion of etiology of stuttering, however, it seems important to note the historical importance of behavioral theories and to emphasize the continued importance of the methods of experimental analysis of behavior in attempts to understand what variables contribute to the development of stuttering and in the systematic evaluation of the procedures used to ameliorate stuttering.

Genetically Transmitted or Acquired Sensorimotor Deficit

The large and ever-growing literature on motor performance and stuttering is contradictory, with some studies indicating stutterers perform more poorly than nonstuttering controls and other studies showing no differences between groups of stutterers and nonstutterers (Andrews et al., 1983; Van Riper, 1982). Such studies have assessed performance in both children and adults, but it appears that we are still far from answering these questions: Is stuttering a speech disorder that arises in part from a

deficit in sensorimotor organization and if there is such a deficit, is it speech specific, with no relation to other motor skills? Resolution of these issues is important if progress is to be made in the attempt to understand stuttering.

It may seem surprising that this section includes the possibility that a sensorimotor deficit could be acquired. The idea that the deficit that produces the coordinative breakdowns of stuttering is genetically transmitted, although not usually directly stated, is often implied. For example, Cox (1988) considered "the possibility that we could actually identify the gene or genes responsible for the transmission of stuttering. Would it be a gene or genes involved in the complex developmental process of hemispheric lateralization? Or perhaps the gene would code for a structural protein in the larynx which is abnormal in persons who stutter and prevents appropriately coordinated communication with speech centers in the brain" (p. 36). At this point, as Cox clearly states, however, we have no idea what is genetically transmitted that is significant for the development of stuttering. An attractive hypothesis for investigation is that a component of stuttering that is genetically transmitted is a "vulnerable" neuromotor system. Equally probable is that the genetically transmitted susceptibility to stutter is some configuration of emotional characteristics that produces degraded performance of a potentially normal motor system. Many other possibilities could be discussed.

It seems important at this point to keep an open mind about what might be genetically transmitted and to recognize that the speech motor learning processes in children who stutter might be strongly influenced by environmental factors. A recent experimental finding suggests that mothers of young stutterers talk faster than mothers of nonstutterers (Meyers & Freeman, 1985b). Speech rate clearly has an effect on fluency (e.g., Starkweather, 1987a). Perhaps with a more difficult model to imitate for production, speech motor learning is disrupted.

Perinatal, Medical, Developmental, and Language Histories

Based on reviews of a number of studies, there is general agreement that there are no differences in perinatal or medical histories of stutterers compared to the general population (Bloodstein, 1981; Van Riper, 1982). In relation to developmental and language histories, Andrews et al. (1983) state, "There is robust evidence that stutterers as a group differ in IQ distribution, are late and poor talkers, have difficulty in stimulus recognition/recall in complex auditory tasks, and lag in tests of sensory-motor response" (p. 232). I would disagree that the evidence is "robust" in all of these cases. In considering potential risk factors for stuttering, however, there appears to be good evidence that an early history of speech or language difficulty besides stuttering may be significant (Bloodstein, 1981). Blood and Seider (1981), on the basis of information on 1,060 stutterers aged 14 or younger, found that 68% of the children had other problems, most frequently articulation and/or language disorders.

An ambitious study by Cox, Seider, and Kidd (1984) attempted to examine the potential role in stuttering of prenatal, medical, developmental, speech, language, educational, and social histories as well as the factors of personality and parent-child interaction. In this study, 14 "high-density" stuttering families (a family that includes at least five individuals who reported or exhibited stuttering for at least 6 consecutive months) and 10 control families with no stuttering members provided case history interview data and completed self-report inventories. Use of the "high-density paradigm" allows comparisons of stutterers and their nonstuttering relatives and of stutterers and their families to individuals from families with no history of stuttering. The logic is that any factors, environmental or genetic, that are significant for the development of stuttering should be highly concentrated in families with many affected members. Cox et al. (1984) found no prenatal, developmental, medical, educational, or social factors that distinguished stutterers or their families. Further, they found no evidence of differences between stutterers, their nonstuttering relatives, and controls on measures of anxiety, familial attitudes toward speech, or parental or child behavior. The lack of any clear support for the presence of common environmental factors among these high-density families suggested to Cox et al. (1984) that there may be heterogeneous etiologies of stuttering. Although the results of this study do not provide any clear clues as to the nature of environmental variables important in stuttering, the method of this study is significant. The use of the high-density paradigm could be pursued to assess whether other characteristics are common to families of stutterers.

Cultural Factors

The literature on stuttering contains an abundance of long-standing debates. Perhaps one of the most colorful is the debate concerning the existence of cultures without stuttering or a word to designate stuttering. This issue was of critical importance to Johnson's (1942) diagnosogenic theory of stuttering, and his students Snidecor (1947) and Stewart (1959) studied two tribes of North American Indians, the Bannock and Shoshone, which were claimed not "to have a word for it." The anthropologist Liljeblad (1967) contested these claims. This issue was revisited recently within a research report by Zimmermann, Liljeblad, Frank, and Cleeland (1983) and a letter from Stewart (1985). Despite Stewart's persistent claims, the weight of evidence suggests that these tribes did in fact have members who stuttered and "words for it." When available data both from early anthropological and later epidemiological studies are pooled, one is impressed with the universality of stuttering (Van Riper, 1982). Stuttering appears in diverse cultures throughout the world, however there is also evidence that the prevalence of stuttering varies between cultures (Bloodstein,

1981; Van Riper, 1982). The data have been interpreted to suggest that competitive societies with high standards of individual achievement have a higher prevalence of stuttering (Bloodstein, 1981).

CONCLUSION: FUTURE DIRECTIONS

Multiple Etiologies?

Two questions directed to the writers in this section were "Are there multiple etiologies of stuttering, and what might these be?" There is clear evidence that multiple factors are involved in the development of stuttering. The suggestion of multiple etiologies invokes the possibility that there are factors that are significant for one individual's path to the diagnosis of stuttering, while for another individual, a completely different set of conditions may lead to the disorder. In view of the heterogeneity of stuttering, this is a plausible way to think about the development of stuttering, but perhaps not the most useful one. This point of view emphasizes differences between stutterers and suggests a focus on types. It also is consistent with developing diverse and narrowly focused theories to account for different "types" of stuttering.

It would be more useful to seek a common set of principles that can explain stuttering in all of its forms. In the last chapter of his book, Van Riper (1982) attempted a synthesis of the literature he so thoroughly reviewed. He rejected the notion that we will ever find a "simple and elegant one-factor explanation for stuttering" and suggested that perhaps one day we could compute an "esoteric formula" that would indicate the probability that a given individual would stutter. For each individual Van Riper's formula would contain different weights assigned to factors such as familial incidence, sex, coordinative ability, proneness to anxiety, reactiveness to communicative stress, intelligence, and language skills. This approach recognizes that one individual may have a very heavy weighting on, for example, a sensorimotor factor and very little weighting on emotional factors associated with stuttering. Another individual may have reversed coefficients on these factors. Nonetheless, both may be equally at risk for being placed in a single diagnostic category, stuttering. Thus, I would argue that, in terms of both practical and theoretical goals, it is more appropriate to think of "multiple risk factors in the etiology of stuttering" rather than "multiple etiologies."

Research Needs and Directions

Two important long-term goals of research on the etiology of stuttering are to develop an index of the risk for a child to become a stutterer, that is, to specify the factors in Van Riper's formula and to delineate the environmental factors associated with the development of stuttering. If a child were known to have a high degree of risk for becoming a stutterer, environmental variables presumably could be manipulated to reduce the probability of the emergence of the disorder. Many intermediate steps are needed in order to pursue these goals. In particular we need to know what factors to include in calculating risk. Following is a list of some experimental questions critical to progress in this area. With each question are general indications of methodology currently available to address these questions.

1. Are there common features measurable in peripheral physiological events associated with stuttering in adults and children? Possible methodologies: EMG and kinematic measures of activity of the respiratory, laryngeal, and articulatory systems; autonomic measures such as peripheral blood flow, skin conductance, and heart rate. If we can determine the degree of association between measures derived from physiological signals and speech breakdown, these measures might be used to assess risk in young children. For example, if oscillation of muscle activity is a common feature of EMGs recorded during speech of stutterers (e.g., Smith, 1989) but is not associated with disfluencies of normal speakers, this would suggest use of a measure of EMG oscillation in assessing risk. This might be an aid to distinguishing disfluencies that are the early sign of stuttering from those that signal the process of normal speech development. Clearly, as Conture (1987) has discussed, there are difficulties to overcome in applying these techniques to very young children, but these are not insurmountable.

2. Are there any characteristics of high-density stuttering families that distinguish them from families with no stutterers? The paradigm of Cox et al. (1984) could be used to assess differences on many factors, such as interruptive behavior and speech rate (Meyers & Freeman, 1985a,b), neuromotor abilities, or personality factors. Results of such experiments would have important implications for deciding what factors increase risk for stuttering and those that could be modified to decrease risk.

3. Is stuttering a disorder specific to the speech production process? Or does stuttering arise from a generalized sensorimotor deficit, such as a general inability to generate an adequate "time plan" for motor behaviors (Kent, 1984)? Methodology: multivariate analyses of performance, including more subtle measures derived from a wide range of motor behaviors. Many previous experiments have involved measures of relatively crude aspects of motor performance on a single task. Because the results of these studies have been contradictory, it seems likely that no single measure of motor performance in stutterers and nonstutterers can consistently discriminate the two groups. Therefore, progress in this area would seem to depend on use of new methodologies. Resolution of this question is important to our understanding of the nature of stuttering, but also may provide unexpected avenues for assessing risk. If stuttering is associated with a generalized sensorimotor deficit, we may be able to detect evidence of a generalized deficit in measures of motor behaviors that occur well before the typical onset of stuttering (e.g., breathing).

4. How do "linguistic" variables contribute to the increased probability of breakdown in stuttering? What are the precise characteristics of the language and articulatory deficits concomitant to stuttering and how do these relate to the onset of disfluent behavior? Methodology: Careful descriptive analyses of the relationship between early disfluency and variables such as syntactic complexity or lexical development (e.g., Bernstein-Ratner & Sih, 1987; Hegde, 1982); studies that combine measures of motor performance with manipulation of linguistic variables (e.g., Peters & Hulstijn, 1987). Progress in this area would be useful in determining whether there are any specific aspects of language that produce a particular "load" on the developing speech production system and in indicating how such variables may have an effect on the speech production process.

Results of investigations in the four areas listed above will provide many different levels of description of behaviors associated with stuttering. It will be important to avoid the mistake of confusing the level of analysis with the level of explanation sought (Zimmermann & Kelso, 1982). By undertaking a behavioral analysis, an investigator does not necessarily embrace the idea that stuttering is a "purely learned" disorder. In addition, the relevance of all of these levels of analysis to understanding stuttering must be recognized, and that no particular level of description is superior to any other (Perkins & Curlee, 1969). Ultimately the task will be to integrate information from all levels of description into a cohesive theory of stuttering, one that is firmly grounded in an understanding of the principles of operation of normal speech production. When the goals of explanation for a theory of stuttering are clearly articulated, the success of any theory in accounting for all of the relevant phenomena can be evaluated.

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REFERENCES

ADAMS, M. (1974). A physiologic and aerodynamic interpretation of fluent and stuttered speech. *Journal of Fluency Disorders, 1,* 35–47.

ADAMS, M. R. (1984). Laryngeal onset and reaction time of stutterers. In R. F. Curlee, & W. H. Perkins (Eds.), *The nature and treatment of stuttering: New directions* (pp. 89–103). Boston, MA: College-Hill Press.

ANDREWS, G., CRAIG, A., FEYER, A. M., HODDINOT, S., HOWIE, P., & NEILSON, M. (1983). Stuttering: A review of research findings and theories circa 1982. *Journal of Speech and Hearing Disorders, 48,* 226–246.

BAUMGARTNER, J. M., & BRUTTEN, G. J. (1983). Expectancy and heart rate as predictors of the speech performance of stutterers. *Journal of Speech and Hearing Research, 26,* 383–388.

BERNSTEIN-RATNER, N., & SIH, C. C. (1987). Effects of gradual increases in sentence length and complexity on children's dysfluency. *Journal of Speech and Hearing Disorders, 52* (3), 278–287.

BLOOD, G. W., & SEIDER, R. (1981). The concomitant problems of young stutterers. *Journal of Speech and Hearing Disorders, 46,* 31–33.

BLOODSTEIN, O. (1960a). The development of stuttering: I. Changes in nine basic features. *Journal of Speech and Hearing Disorders, 25,* 219–237.

BLOODSTEIN, O. (1960b). The development of stuttering: II. Development phases. *Journal of Speech and Hearing Disorders, 25,* 366–376.

BLOODSTEIN, O. (1981). *A handbook on stuttering (3rd edition).* Chicago: National Easter Seal Society.

BROWN, S. F., & HULL, H. C. (1942). A study of some social attitudes of a group of 59 stutterers. *Journal of Speech and Hearing Disorders, 7,* 323–324.

BRUTTEN, E. J., & SHOEMAKER, D. J. (1967). *The modification of stuttering.* Englewood Cliffs, NJ: Prentice-Hall.

CHMELOVA, A., KUJALOVA, V., SEDLACKOVA, E., & ZELANY, A. (1975). Neurohumorale Reaktionen bei Stotterern und Polterern. *Folia Phoniatrica, 27,* 283–286.

CONTURE, E. G. (1987). Studying young stutterers' speech productions: a procedural challenge. In H. F. M. Peters, & W. Hulstijn (Eds.), *Speech motor dynamics in stuttering* (pp. 117–140). New York: Springer-Verlag.

COSTELLO, J. M. (1984). Treatment of the young chronic stutterer. In R. F. Curlee, & W. H. Perkins (Eds.), *Nature and treatment of stuttering: New directions* (pp. 187–214). Boston MA: College-Hill Press.

COX, N. J. (1988). Molecular genetics: The key to the puzzle of stuttering? *American Speech-Language and Hearing Association, 30,* 36–40.

COX, N. J., SEIDER, R. A., & KIDD, K. K. (1984). Some environmental factors and hypotheses for stuttering in families with several stutterers. *Journal of Speech and Hearing Research, 27* (4), 543–548.

EDGREN, B., LEANDERSON, R., & LEVI, L. (1970). A research programme on stuttering and stress. *Acta Otolaryngologica, 263,* 113–118.

HAMRE, C. E. (1984). Stuttering as a cognitive-linguistic disorder. In R. F. Curlee, & W. H. Perkins (Eds.), *Nature and treatment of stuttering: New directions* (pp. 237–259). Boston MA: College-Hill Press.

HARRINGTON, J. (1988). Stuttering, delayed auditory feedback, and linguistic rhythm. *Journal of Speech and Hearing Research, 31,* 36–47.

HEGDE, M. N. (1982). Antecedents of fluent and dysfluent oral reading: A descriptive analysis. *Journal of Fluency Disorders, 7,* 323–341.

HOWIE, P. M. (1981). Concordance for stuttering in monozygotic and dizygotic twin pairs. *Journal of Speech and Hearing Disorders, 24,* 317–21.

ICKES, W. K., & PIERCE, S. (1973). The stuttering moment: A plethysmographic study. *Journal of Communication Disorders, 6,* 155–164.

JAYARAM, M. (1984). Distribution of stuttering in sentences: Relationship to sentence length and clause position. *Journal of Speech and Hearing Research, 27,* 338–341.

JOHNSON, W. (1942). A study of the onset and development of stuttering. *Journal of Speech and Hearing Disorders, 7,* 251–257.

JOHNSON, W. (1955). A study of the onset and development of stuttering. In W. Johnson, & R. R. Leutenegger (Eds.), *Stuttering in children and adults.* Minneapolis: University of Minnesota Press.

KENT, R. D. (1984). Stuttering as a temporal programming disor-

der. In R. F. Curlee, & W. H. Perkins (Eds.), *Nature and treatment of stuttering: New directions* (pp. 283–301). Boston MA: College-Hill Press.

LILJEBLAD, S. (1967). *The Indians have a word for it.* Paper presented to the Nevada State Speech Association.

MACKAY, D. G., & MACDONALD, M. C. (1984). Stuttering as a sequencing and timing disorder. In R. F. Curlee, & W. H. Perkins (Eds.), *The nature and treatment of stuttering: New directions* (pp. 261–282). Boston, MA: College-Hill Press.

MARTIN, I., & VENABLES, P. H. (1980). *Techniques in psychophysiology.* New York: John Wiley & Sons.

MARTIN, R. R., & HAROLDSON, S. K. (1988). An experimental increase in stuttering frequency. *Journal of Speech and Hearing Research, 31,* 272–274.

MEYERS, S. C., & FREEMAN, F. J. (1985). Interruptions as a variable in stuttering and disfluency. *Journal of Speech and Hearing Research, 28,* 428–435.

MEYERS, S. C., & FREEMAN, F. J. (1985). Mother and child speech rates as a variable in stuttering and disfluency. *Journal of Speech and Hearing Research, 28,* 436–444.

NEILSON, M. D., & NEILSON, P. D. (1987). Speech motor control and stuttering: A computational model of adaptive sensory-motor processing. *Speech Communication, 6,* 325–333.

PERKINS, W. H., & CURLEE, R. F. (1969). Causality in speech pathology. *Journal of Speech and Hearing Disorders, 34,* 231–238.

PETERS, H. F. M., & HULSTIJN, W. (1984). Stuttering and anxiety: The difference between stutterers and nonstutterers in verbal apprehension and physiologic arousal during the anticipation of speech and non-speech tasks. *Journal of Fluency Disorders, 9,* 67–84.

PETERS, H. F. M., & HULSTIJN, W. (1987). Programming and initiation of speech utterances in stuttering. In H. F. M. Peters, & W. Hulstijn (Eds.), *Speech motor dynamics in stuttering* (pp. 185–195). New York: Springer-Verlag.

PETERS, H. F. M., & HULSTIJN, W. (1987). (Eds.). *Speech motor dynamics in stuttering.* New York: Springer-Verlag.

PRINS, D. (1972). Personality, stuttering severity, and age. *Journal of Speech and Hearing Research, 15,* 148–154.

SHEEHAN, J. G. (1953). Theory and treatment of stuttering as an approach-avoidance conflict. *Journal of Psychology, 36,* 27–49.

SHEEHAN, J. G. (1970). *Stuttering: Research and therapy.* New York: Harper & Row.

SMITH, A. (1989). Neural drive to muscles in stuttering. *Journal of Speech and Hearing Research, 32,* 252–264.

SNIDECOR, J. C. (1947). Why the Indian does not stutter. *Quarterly Journal of Speech, 33,* 493–495.

STARKWEATHER, C. W. (1987a). Laryngeal and articulatory behavior in stuttering: Past and future. In H. F. M. Peters & W. Hulstijn (Eds.), *Speech motor dynamics in stuttering* (pp. 3–18). New York: Springer-Verlag.

STARKWEATHER, C. W. (1987b). *Fluency and stuttering.* Englewood Cliffs, NJ: Prentice-Hall.

STEWART, J. L. (1985). Stuttering Indians: A reply to Zimmermann et al. *Journal of Speech and Hearing Research, 28,* 313–315.

STROMSTA, C. (1987). Acoustic and electrophysiologic correlates of stuttering and early developmental reactions. In H. F. M. Peters, & W. Hulstijn (Eds.), *Speech motor dynamics in stuttering* (pp. 267–277). New York: Springer-Verlag.

VAN RIPER, C. (1982). *The nature of stuttering (2nd edition).* Englewood Cliffs, NJ: Prentice-Hall.

WEXLER, K. B. (1982). Developmental disfluency in 2-, 4-, and 6-year-old boys in neutral and stress situations. *Journal of Speech and Hearing Research, 25,* 229–234.

WINGATE, M. (1964). Standard definition of stuttering. *Journal of Speech and Hearing Disorders, 29,* 484–489.

WINGATE, M. E. (1962). Personality needs of stutterers. *Logos, 5,* 35–37.

WISCHNER, G. J. (1952a). Anxiety-reduction as reinforcement in maladaptive behavior: Evidence in stutterers' representations of the moment of difficulty. *Journal of Abnormal Social Psychology, 47,* 466–571.

WISCHNER, G. J. (1952b). An experimental approach to expectancy and anxiety in stuttering behavior. *Journal of Speech and Hearing Disorders, 17,* 139–154.

WOODGER, J. H. (1952). *Biology and language.* Cambridge MA: Cambridge University Press.

YAIRI, E. (1983). The onset of stuttering in two- and three-year-old children: A preliminary report. *Journal of Speech and Hearing Disorders, 48* (2), 171–177.

ZIMMERMANN, G. N. (1980). Stuttering: A disorder of movement. *Journal of Speech and Hearing Research, 23,* 122–136.

ZIMMERMANN, G. N., & KELSO, J. A. (1982). Remarks on the "causal" basis of stuttering. *The Proceedings of Van Riper Lecture Series.*

ZIMMERMANN, G. N., LILJEBLAD, S., FRANK, A., & CLEELAND, C. (1983). The Indians have many terms for it: Stuttering among the Bannock-Shoshone. *Journal of Speech and Hearing Research, 26,* 315–318.

ZIMMERMANN, G. N., SMITH, A., & HANLEY, J. M. (1981). Stuttering: In need of a unifying conceptual framework. *Journal of Speech and Hearing Research, 46,* 25–31.

D. SUBTYPING STUTTERERS

Chapter 6

SUBTYPING CHILD STUTTERERS FOR RESEARCH PURPOSES

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CURRENT KNOWLEDGE

Stuttering Typology And Its Impact

The literature on stuttering includes many detailed descriptions of a large variety of both overt and covert characteristics or "symptoms" of the disorder (e.g., Bloodstein, 1961; Froeschels, 1923; Van Riper, 1971). Not only is the overall number of characteristics large, but the variants of each one and their peculiar combinations as expressed through individual stuttering patterns is apparently limitless. No stutterer exhibits all possible symptoms; some may not exhibit even common ones (Barr, 1940). Despite this apparent heterogeneity, experts and lay people alike have shown an undeniable inclination to refer to all the variants as "stuttering." From the outset then, the task of identifying characteristics which can meaningfully distinguish among types or subgroups of stutterers poses a challenge.

The idea that stutterers can be classified according to types has been expressed by numerous writers during the past several hundred years. In a review of the literature, Van Riper (1971) has traced early attempts of stuttering typology as far back as the 16th century. A more recent review of the subject was included in Preus' work of 1981. Such typologies, often speculative in nature, represent several frames of reference.

Etiology has been a frequent basis of classification. This is well-illustrated by Luchsinger and Arnold's (1965) six-type scheme which includes organic (inherited), symptomatic (of organic lessions), developmental, traumatic, physiological, and hysterical stutterers. St. Onge (1963) suggested three types: organic, psychogenic, and speech phobic stutterers. Brill's (1923) psychologically based classification and Canter's (1971) neurogenic types of stuttering provide additional examples.

A second approach to classification has been based on a prominent stuttering characteristic (symptom) or behavior of the stutterers. Such was the distinction between clonic and tonic stutterers (Froeschels, 1943), interiorized and exteriorized stutterers (Douglass & Quarrington 1952), stutterers who exhibit different adaptation effects (Newman, 1963) or different formant transition in the acoustic spectrum of their speech (Stromsta, 1965).

Still another approach was employed by investigators using certain characteristics of the stutterer such as sex (Silverman, 1986), familial history of stuttering (Seider, Gladstein, & Kidd, 1982; 1983) and ear preference during dichotic listening tasks (Hinkle, 1971) as the basis for classification. Finally, multiple-factor approaches have been used based on combinations of variables pertaining to the characteristics of stutterers and/or stuttering symptomatology (Andrews & Harris, 1964; Van Riper, 1971). Within this general category, Schwartz and Conture (1988) restricted their approach to objective, identifiable measures of speech and nonspeech behaviors.

In spite of the diversity in stuttering manifestations and a number of references to typology in the literature, the overwhelming attitude among experts, especially researchers, has been to approach stuttering as a pathognomonic monolith (St. Onge, 1963; St. Onge & Calvert, 1964). In countless studies and clinical programs aimed at adults and children, subjects were indiscriminately included simply because they were labeled "stutterers" and assumed to exhibit a single disorder. Ambiguity in the outcome of this work may be the direct effect of a long-term reluctance to consider subgroups.

Research Concerning Subgroups

The failure to consider heterogeneity among subjects in stuttering research can be partially explained by the scant, relatively late, experimental, or other evidence concerning type or subgroup differentiation according to any of the systems mentioned above. In adults, the first comprehensive investigation of the subgroup hypothesis was conducted by Berlin in 1954 comparing seven etiological groups of adult subjects defined prior to the investigation. Interestingly, the only subgroup that emerged with some distinction, the one presumably with brain damage, evidenced a more gradual onset of stuttering.

A few additional studies of differences among adults appeared since that time concerning psychodynamic processes and stuttering symptoms. Emerick (1966) found essentially no differences between tonic and clonic stutterers on all three psychological correlates: responses to frustration, level of aspiration, and verbal intelligence. Kroll (1976) had only limited success substantiating a predetermined classification of subjects as interiorized and exteriorized stutterers. The two subgroups differed on only three (of seven) psychosocial and speech variables: communicative goals, group affiliation, and awareness.

Based on neural organization as revealed in listening tasks, Hinkle (1971) found that scores for ear preference in dichotic listening were successful in differentiating subgroups in terms of unique speech patterns. Seider, Gladstein, and Kidd (1983) considered subtyping stutterers on the basis of the influence of familial history of stuttering on spontaneous recovery. They reported that the rate of reported spontaneous recovery in stuttering relatives of persistent adult stutterers fell within the recovery range for unselected stutterers. However, their data showed sex to be a significant variable in recovery. Overall recovery among stuttering female relatives was significantly higher and occurred at an earlier age than recovery among male relatives. Also, the probands' (the subjects through whom relatives were identified) opposite-sex siblings were more likely to recover from stuttering than the same-sex siblings.

Focusing on observable and quantifiable features of stuttering, Prins and Lohr (1972) correlated 46 visible or audible phenomena that occurred during stuttering moments of adult subjects. Their statistical analyses yielded 10 factors including severity, tension, adaptation, and lateralization of behaviors such as jaw and lip deviations. The investigators suggested that factor scores or profiles might be useful in subgrouping stutterers. Although they did not test out this assumption, their work provided the impetus for future research that was applied to the classification of child stutterers.

Systematic research on types of child stutterers began with Andrew and Harris' (1964) study of school-age children. Multiple regression analyses were employed to identify differences on any of 37 variables such as age, intellectual level, age at stuttering onset, and anxiety when each of three major variables: sex, severity of stuttering, and familial history of stuttering, was used as a criterion for group comparisons. Significant differences were found only between male and female stutterers on several items labeled as "neurological speech competence."

Andrew and Harris (1964) also applied factor analysis techniques to their data. These statistical procedures revealed three possible factors (subgroups). Factor I was composed of items reflecting poor intellectual abilities, late and abnormal speech development, and mother's failure to cope. Earliest symptoms were mild repetitions which did not change much later on. This factor had striking resemblance to features that differentiated stutterers from nonstutterers (lack of capacity to think, talk, and behave). Factor II included items concerned primarily with severe stuttering. It described children with poor speech, early onset of stuttering and disfluency patterns that started with repetitions but developed into complex, severe blocks, and prolongations. The children also tended to be overactive and irritable. Children classified in Factor III (which accounted for only a small proportion of the variance) were described as intelligent, coming from a neurotic home environment, and exhibiting anxiety-aggressiveness behaviors. They had developed a mild form of stuttering.

As mentioned earlier, Schwartz and Conture (1988) identified subgroups of stutterers by analyzing the observable type of stuttering produced (sound or syllable repetitions and sound prolongations) and 14 speech/nonspeech behaviors associated with the stuttering. Their 43 subjects ranged in age from 3:10 to 9:4 with a mean age of 5:11. The 14 behaviors were condensed to form three indices: (1) Sound Prolongation Index (SPI), the ratio of sound prolongations to the total number of stutterings; (2) Nonspeech Behavior Index (NBI), the average number of nonspeech behaviors per stuttering; and, (3) Behavioral Variety Index (BVI), the average number of different types of behaviors per stuttering. Classification of subjects using cluster analysis of the three indices revealed five subgroups that varied in their scores on the these indices. The investigators suggested that the five subgroups could possibly be reduced to only two: young stutterers who predominantly exhibit repetitions and those who predominantly exhibit sound prolongations. They concluded that group similarities and differences in terms of disfluency type and associated behaviors might become an important issue in the study of the onset and development of stuttering.

Some support to the validity of these subgroups was provided by Schwartz (1987), who compared 15 of the same young stutterers on measurements of rib and abdomen displacement, lip closing and opening, and the onset of vocal fold contact during stuttering. While all subjects showed the same sequence of muscle activity, significant differences among the clusters were found in the timing relationships among the three physiological systems.

Perhaps the best known work concerning subgroups of stuttering children was reported by Van Riper in 1971 summarizing case reports of longitudinal observations of 44 young stutterers. He distinguished four subgroups having different combinations of age at stuttering onset, manner of onset, symptomatology, and general speech skills. Most significantly, his subgroups exhibited different developmental "tracks" and prognoses:

Track I (50%) had an early stuttering onset (ages 2 to 3) following a period of normal speech and language development. The early characteristic was easy syllabic repetitions. Later on, sound prolongations, physical tension, and strong emotional reactions became predominant. This group had the highest rate of spontaneous recovery and the most favorable prognosis in therapy.

Track II (25%) has later onset than Track I, often coinciding with the appearance of first sentences. Articulation and language were delayed. Early symptoms

were hurried unorganized syllabic repetitions. The overall speech was unorganized giving the impression of cluttering. Sound prolongations and physical tension were rare and emotional reactions were low-level. There was little change in the symptomatology over time.

Track III (12%) had sudden and usually late onset (ages 5 to 9) against the background of normal articulation/language development. Predominant symptoms were tonic blocks and sound prolongations, physical tension and its secondary characteristics and emotional reactions. This group developed the most morbid form of stuttering with the most intense emotional reactions.

Track IV (13%) had sudden and late onset with no previous history of speech/language problems. The predominant symptom was highly stereotyped word and phrase repetitions or pauses accompanied by stereotyped postures (e.g., open jaw). There was little emotional reaction and almost no change in symptomatology over time.

Van Riper's work established the importance of developmental features in stuttering typology. Although his report was based on retrospective analyses of clinical files and lacked quantifiable, objective data, the usefulness of his track system for classifying stutterers has received support in two investigations. Daly (1981) used data from files and video tapes of 138 stutterers ages 8 to 20 and was able to assign 83% of them to the first three tracks only with a reasonably similar distribution (54%, 24%, and 5% respectively) to Van Riper's. His sample, however, included only five females and excluded subjects in the early childhood ages. An interesting finding was that large percentages of subjects in Track II had motor and reading problems in addition to the typical delayed speech development. Comparisons of pre- and post-therapy stuttering severity scores also indicated differences among the subgroups in response to therapy. With only part of the data subjected to statistical analyses, it was concluded that Track II subjects had significantly poorer progress through therapy than subjects in Track I.

The second investigation that bears on Van Riper's track system was reported by Preus (1981) who gathered data on 70 speech and psychosocial variables for 100 stutterers ages 16 to 22 years. Although his subjects were too old for direct comparisons with children, it is interesting to note that Preus' forced classification of all subjects into the four tracks resulted in a generally similar distribution (58%, 18%, 23%, and 1% respectively) to those reported by Van Riper (1971) and Daly (1981). Multivariate analyses revealed that only Track II was significantly different from the rest of the sample. Variables that contributed most to the contrast indicated the presence of cluttering, articulation disorders, delayed language, and relatively mild stuttering characterized by more repetitions and fewer prolongations.

The conclusions of Daly (1981) and Preus (1981) regarding the differentiating role of additional speech-language difficulties in young stutterers were amplified by two other reports. Blood and Seider (1981) suggested that subgroups of school-age stutterers could possibly be formed based on the presence or absence of concomitant problems. Their survey of speech-language pathologists in the public schools revealed that 32% of all the young stutterers were thought to be free of any other problems, 44% had one concomitant problem, and 24% had two or more such problems. Disorders of articulation were the most common concomitant problem. Becker et al. (1977), cited by Preus (1981) reported a series of studies in Eastern Europe in which he classified stutterers according to the presence or absence of symptoms of organic brain damage. The two main subgroups that he identified in samples of children and adults were (a) the "brain damaged" and, (b) the "neurotic." Subjects in the first subgroup (40% to 80%) began stuttering with the onset of speech, had slow or stationary development of stuttering, showed symptoms of cluttering, dysarthria and other problems. This is a very similar description to Van Riper's Track II type.

Different findings concerning the possible role of speech development in subtyping stutterers were reported by Seider, Gladstein, and Kidd (1982). They examined reports on the time of language onset and the frequency of speech and language problems in a large group of stutterers and their nonstuttering siblings. No differences were found between stutterers and their same-sex nonstuttering siblings or among stutterers grouped according to sex, familial history of stuttering, and recovery from stuttering.

Lastly, the use of the developmental principle in subtyping young stutterers can also be detected in the work of Dostalova and Dosuzkov (1966). These investigators viewed most childhood stuttering as neurotic. Apparently using age at onset as the main criterion for differentiating their 2,000 stutterers, the investigators established three subgroups: *Balbutis Praecox* (39.95%) was observed between age 1 1/2 and the end of the 4th year of life, *Balbtuis Vulgaris* (50.60%) between ages 5 and 8, and *Balbutis Tarda* (9.45%) up to age 13. The differences in the three subgroups was reflected in their responses to therapy. Therapeutic success was reported to be inversely related to age, decreasing from 67.8% to 60.7% to 37.5% in the three groups, respectively. The rate of spontaneous recovery followed a similarly declining curve.

GENERAL RESEARCH NEEDS

The diametrically opposite prospects with which young stutterers are faced, that is, spontaneous remission on one hand and developing a serious communication disorder on the other, present a compelling case for research concerning subtypes of stuttering in early childhood. Although its theoretical significance may be quite apparent, there are also important practical considerations. Investigating subgroups should provide an information base that will assist in the early identification of transient and chronic stutterers, the type and timing of proper treatment, and prognosis for therapy. In short, the ability to make a reliable prediction in each individual case is perhaps the most pressing need as far as young

stuttering children are concerned. Obviously, establishing meaningful subgroups at the very early stages of the disorder will have far reaching influence on the entire approach to stuttering treatment and research.

Although a modicum of research has been undertaken and few experts have expressed the view that stutterers can be differentiated into subgroups, the matter has not been studied in depth. While studies concerned with adults produced only limited positive findings, studies of child subgroups have been considerably more encouraging. Another significant conclusion from the review of the literature is that a search for a single-factor typology, whether etiological or symptomatological, is not likely to be rewarding. Rather, initial findings suggest that future descriptive studies should include a wide range of speech, developmental, and other factors in the data collection process if meaningful subgroups are to be identified.

In searching further for subgroups, several potentially important elements seem to have emerged. These include sex, the age at stuttering onset, the manner of onset (sudden/gradual), the initial complexity/severity of stuttering pattern, intellectual functioning, articulatory proficiency, and the presence of other concomitant problems. Among the latter, the review pointed out cluttering, indications of brain damage, and hyperactivity as deserving extra attention.

The growing evidence of a genetic factor presence in a large number of stutterers suggests that it should be included in future research on subgroups. Although problems with accuracy of such data are recognized, many investigations of familial incidence have clearly shown that the risk for stuttering among relatives of stutterers is markedly elevated above that of control subjects. A good review of this information was published by Sheehan and Costley in 1978. Andrews et al. (1983), estimated that such a risk for first-degree relatives is greater than three times that in the general population. In addition to confirming high familial concentration, extensive investigations by Kidd and his associates (Kidd, Kidd, & Records, 1977; Kidd, Heimbuch, & Records, 1981; Kidd, 1984) also indicated a considerably higher risk for relatives of female than male stutterers, a strong tendency for vertical transmission of stuttering, and sex ratio (favoring males) among affected relatives that was similar to the ratio among probands stutterers.

Further support for the inclusion of the genetic factor in research on stuttering types is derived from a recent publication by Comings and Comings (1987) regarding high incidence of stuttering in other genetic disorders such as the Tourette Syndrome, and these investigators' estimate that between 10% and 20% of all stutterers may carry the TS gene. Also intriguing is the possibility that many young stutterers who exhibit concomitant problems, often of neurogenic nature, represent a subgroup related to the TS gene.

My overall conclusion from the review of the literature is that the prospects for subgrouping of young stutterers are closely tied to comprehensive knowledge of the development of stuttering. Unfortunately, 60 years of extensive research has left such knowledge about stuttering noticeably deficient. The relatively little research on children in general (Adams, 1986) and the cross-sectional nature of much of the current developmental information have been the main reasons for this undesirable state of the art. The view to be advocated here is that large-scaled longitudinal research concerning developmental aspects of early childhood stuttering should constitute a major direction in future research efforts designed to identify subgroups. Specifically, such research should focus on the onset, symptom development, and remission of stuttering.

SPECIFIC SUGGESTIONS FOR RESEARCH

Onset of Stuttering

The question of how and when children first begin to stutter has both theoretical and practical relevance. Froeschels' (1921) and Bluemel's (1932) assertion that stuttering invariably begins with repetitions of syllables and short words devoid of effort and tension, created a rather stereotypic view of the onset of the disorder. These two writers apparently exerted considerable influence on Johnson et al. (1959), who also conceptualized the onset of stuttering as a uniform set of events, always characterized by gradual appearance of mild repetitions. Similar descriptions that failed to recognize other characterizations of onset were provided by Andrews and Harris (1964) and Morley (1957). Only recent studies (Van Riper, 1971; Yairi, 1983) reported substantial variations in onset.

Future research should reevaluate the whole concept of the "stuttering onset" as an identifiable event. It is extremely difficult to tell at what point stuttering, as a speech disorder, truly "begins," and whether or not the child's speech contained abberrations prior to the perceived abnormal disfluencies. Thus, the role and weight of parent and experimenters judgments of stuttering should be reassessed and the usefulness of other means to establish onset determined.

Even if the current perceptually based notion of onset holds, investigators of the phenomenon must cope with the challenge of defining "stuttering" and "stutterer" at the time when such definitions are most difficult to make because of the frequent mild or inconsistent characteristics and their extensive overlap with normal behaviors. Current schemes for diagnosing incipient stuttering (e.g., Adams, 1977; Pindzola & White, 1986) are not based on adequate age-specific and updated norms. Minute differences in criteria may result in discounting many beginning young stutterers. For example, Adams (1977) suggested that the occurrence of at least three iterations of the unit being repeated is a sign of incipient stuttering. Yairi and Lewis (1984), however, reported that the presence of a second repetition unit is the most powerful discriminant between the disfluencies of young stutterers

near the onset of their stuttering and disfluencies of normally speaking children.

To obtain reliable descriptive data of stuttering onset, future investigators should pay careful attention to the age of the subjects. First, they should consider the fact that at least 50% of all onset takes place before age 4 (Andrews, 1984). In the experience of the present writer the percentage is even higher with the largest concentration of onsets under age 3. Therefore, reaching down to lowest possible ages is a critical requirement.

A related consideration is the age-range. For example, the 7-year span between ages 2 and 9 employed in the Iowa Studies (Johnson et al., 1959) is too large, extending beyond reasonable limits of early childhood. When children of this age-range are grouped together and their data averaged, potential elements for differentiating stuttering subgroups such as disfluency characteristics, articulatory and language skills, motor skills, as well as features of the onset itself, are obscured. Thus, discrete age groups should be employed. Dostalova and Dosuzkov's (1966), Van Riper's (1971), and others' emphasis on age at onset in regard to subgrouping, as well as the demonstrated relationship between disfluency and age during the early years of life (Davis, 1939; Yairi, 1982) provide support to the present recommendation. Although Schwartz and Conture (1988) used a wide age-range of subjects and did not show age to be a significant variable in forming subgroups, the authors did not address the issue of stuttering onset and did not include many of the age-sensitive variables mentioned above that might sharpen differentiation.

Timing of observation is another procedural consideration. Because it is virtually impossible to conduct direct observations at the time and place when stuttering first appears, parents remain a valuable source of information concerning the onset of stuttering. However, the lengthy 18-month average interval between stuttering onset and parent interviews in the Johnson et al. (1959) study was a potential source of inaccuracies in their data. To increase reliability and validity of parental reports, they should be obtained in close temporal proximity to the occurrence of onset.

More emphasis should be placed on direct observations of the child's speech and other behaviors rather than on retrospective information of the kind that dominated past research (Glasner & Rosenthal, 1957; Johnson et al., 1959). Therefore, researchers should strive to obtain recordings of speech and test other parameters within a few weeks, or even days, from the time of onset. With proper attention given to referral sources this goal can be achieved for many children.

We should also seek data on the normalcy of the child's speech prior to any reported stuttering. This, of course, is a substantial undertaking that requires recording of speech when children are just beginning to talk at age 1 or 1.5. Because only 5% of the children will eventually exhibit stuttering, such an approach necessitates very large initial subject pools. Fewer subjects will be required by focusing on children with high risks for stuttering, that is, those coming from families with heavy concentration of stutterers. Keeping in mind the constraints of such selective data, important information can be gained.

The usefulness of several of the above suggestions has been demonstrated by the present author. When the interval between stuttering onset and parent interview was shortened to only 6 months (1/3 of previous research), and subjects' age varied from 24 to 48 months (Yairi, 1983, Yairi & Ambrose, 1987), the substantial variations observed in the onset and early stuttering characteristics were contrary to conventional views. For example, (a) the mean age at onset was as early as 28 months, (b) girls had earlier onset than boys, (c) the sex ratio was nearly equal, (d) about 36% of the subjects were reported to have sudden onset, (e) more girls than boys had sudden onset, (f) most parents perceived early stuttering to be associated with some tension (only 32% reported simple, easy repetitions), and (g) 18% of parents reported secondary physical characteristics such as facial grimaces. Further, speech samples recorded from 2- and 3-year-old children near the onset of stuttering revealed marked differences in frequency/type of disfluency (Yairi & Lewis, 1984) and in the patterns of spatial proximity (clustering) of disfluency (Hubbard & Yairi, 1988) when compared to nonstuttering children. Such differences were not recognized in previous research (e.g., Johnson et al., 1959).

Development and Remission

Knowledge of the course of any disorder is basic to the understanding of that disorder. Although it has been widely recognized that substantial changes in the characteristics of stuttering occur as the disorder persists, the nature, timing, and dynamics of these changes have been scarcely investigated. Much of the present documentation is based on general summaries of clinical impressions or analyses of old clinical files (e.g., Bloodstein, 1961; Froeschels, 1943; Van Riper, 1971), not on firsthand, quantified, and reliable data collected through controlled methods. Only limited data are available on the changes that occur over time in basic characteristics of disfluency. There are only minimal data concerning the appearance and changing character of awareness of stuttering in children (Dixon, 1965; Giolas & Williams, 1958; Yairi, 1983), a concept that constitutes a cornerstone of both stuttering theory and therapy. Similar statements can be made concerning emotional reactions to stuttering, "secondary" characteristics (physical tensions reactions associated with disfluencies), other "symptoms" such as pitch breaks, vocal fry and word substitution, or concerning properties of fluent speech.

Overt physical tensions and other associated behaviors provide a good example of the gaps in our information. Typically, these secondary characteristics or "symptoms" have been regarded as a relatively late developing phenomena, reflecting growing emotional and struggle reactions. Yairi (1983), however, reported on parent recollections of secondary characteristics being present at the beginning of the stuttering problem of several young

children. Schwartz, Zebrowski, and Conture (1986) and Schwartz and Conture (1988) provided objective evidence of secondary characteristics in young stutterers that were derived from video recordings.

No less surprising is the fact that the phenomenon of spontaneous recovery, estimated to occur in between 32% and 79% of stutterers (Andrew, 1984; Johnson et al., 1959), has not been dealt with adequately. Much of the current information concerning spontaneous recovery was generated through retrospective studies of questionable validity and reliability (Dixon, 1965; Milisen & Johnson, 1936; Sheehan & Martyn, 1970), or nonquantified observations by researchers (Andrews & Harris, 1964; Dostalova & Dosuzkov, 1966; Johnson et al., 1959). These investigators failed to support subjective impressions with objective speech-based or nonspeech data. In many surveys, elementary scientific procedures such as definitions of "stuttering" or "recovery" were absent, inviting skeptical evaluation of their validity (Ingham, 1976; Young, 1975). Not only has the precise percentage of recovery remained unclear but also its timing and stability have not been determined. Furthermore, there is virtually no credible knowledge concerning factors that govern, or are associated with, the occurrence of recovery.

In spite of the many questions concerning the stuttering symptom formation as well as the amelioration of the disorder, and the indications in the literature of possible relationships between developmental trends and subtypes of stutterers, the obvious need for longitudinal studies in children has not been met. Recent investigations based on longitudinal methodology (Fritzell, 1974; Ohashi, 1973; Ryan, 1984; Yairi & Ambrose, 1987) signal an encouraging change in interest but they were limited in scope and procedures. Although not excluding other research, the present author believes that longitudinal studies have the best potential for providing (a) accurate accounts of the long-term changes in stuttering and (b) valid data on the relationships of such changes to a host of variables in the stutterer and his/her environment. Only through continuous follow-ups of the same subjects can the intricate dynamics of the development of stuttering be established.

The first stage in longitudinal studies should concentrate on the onset of stuttering, providing extensive case histories and early baselines for disfluency and other measures. Initial contact, recording, and testing should be conducted *immediately* upon referral. In my experience, about 10% of the sample may be lost to spontaneous recovery within a month, even when children exhibit unquestionable stuttering characteristics. Such losses would have a substantial influence on the data and its interpretation.

The longitudinal study should extend over a 10-year period or longer. It is important to extend the study until after siblings of subjects pass age 8 (the point where 95% of the risk for developing stuttering is over). Attention to this aspect will increase the accuracy of data on the incidence of stuttering among siblings. Also, because of predictable loss of subjects due to spontaneous recovery, as well as usual attrition over the years, the initial subject pool should be large, consisting perhaps of several hundred stutterers.

For the duration of the study, the same stuttering children who participated in the first stage should be reevaluated and retested periodically. In my experience, three or four reevaluation sessions per year should be scheduled during the first 2 years with a reduced schedule during succeeding years.

At every testing period, data pertaining to a wide range of variables should be obtained. These include indices of disfluency, associated secondary characteristics (the use of video recording is highly recommended), overall stuttering severity scores, acoustical features of disfluencies, acoustical features of fluent speech, awareness of stuttering, emotional reactions to stuttering as well as general anxiety reactions, neuromotor skills, several measures of language skills, phonological skills, social maturity, intellectual abilities, hearing, behavioral problems, health status, and more. Systematic recording of parents' interim progress reports are also important to evaluate the short-term stability or cycles in the phenomena of stuttering.

Longitudinal studies of preschool children can be planned to test the existence of subgroups defined *a priori* as well as identify subgroups on an *a posteriori* basis. The former alternative provides the necessary framework for meaningful data collection and there are reasonable grounds to suggest relevant hypotheses concerning *a priori* subgroups of young stutterers.

Groups can be established according to a complex of factors including age at onset, patterns of onset, patterns of overt characteristics, articulation-language proficiency, and presence of other concomitant problems. For example, based on the literature reviewed, it might be hypothesized that subjects exhibiting early and gradual onset of stuttering, an initial dominant overt symptom of clonic-type disfluencies (sound and syllable repetitions), normal articulation, and no concomitant problems will develop a milder form of stuttering and be more likely to recover than subjects exhibiting sudden onset, a dominant symptom of tonic-type disfluencies (sound prolongations), delayed speech, or other concomitant problems.

Another hypothesis is that stuttering development and recovery is related to gender. The increasing sex ratio with age, the differences between boys and girls in age at onset, and some differences in severity (Yairi & Ambrose, 1987) seem to predict that female stutterers will be more likely to recover than male stutterers. No clear hypothesis is possible at this point as to whether or not stutterers with no or only light "genetic loading" have better chances to recover than stutterers with heavy loading. In light of the previous discussion, however, such comparisons should be pursued.

Longitudinal studies of selected children born to families with high density of stuttering incidence (Cox, Seider, & Kidd, 1984) may be of special interest. In fact, those children should be periodically observed, recorded, and tested long before onset of stuttering.

Several *a posteriori* analyses are possible. Close inspection of individual longitudinal profiles displaying

changes in the various measures across several years is a good starting point. By following additions, deletions, and consistency in the various measures, much insight can be gained regarding the dynamics of stuttering, different tracks along which the disorder develops, and the emergency of subgroups. Multifactorial statistical methods such as regression analysis and cluster analysis should then be applied to evaluate the possible relationships of the hundreds of information items that can be collected in a longitudinal study. Such techniques appear to have been rather useful in differentiating subgroups of stutterers (Andrews & Harris, 1964; Schwartz & Conture, 1988).

One difficult problem inherent in longitudinal studies of stuttering concerns the provision of parent counseling and/or speech therapy for the children. Clinical intervention may alter symptom development and contaminate data on spontaneous recovery. Withholding therapy purposely, on the other hand, presents a serious ethical dilemma. Undoubtedly, this was an important reason why longitudinal research has not been an attractive approach. Although the ethical issue still presents a "roadblock" to future research, there are some practical alternatives for dealing with this problem.

First, our own experience has been that there are always parents who are either not interested or unable to provide therapy for their children. Another group consists of parents who assume, at least temporarily, a "wait and see" attitude, often on the advice of their physician. They fear that therapy will worsen the child's stuttering and prefer to delay it. In other words, it can be expected that, by choice, a good number of children will not receive therapy, at least during the initial part of the study.

Another alternative is to provide a standard counseling to parents of all children at the beginning of the study deferring direct speech therapy for 1 to 2 years. It is possible to assume this position on the grounds that there is no experimental evidence indicating advantages of direct therapy for preschool stutterers compared to parent counseling. It is also possible to conduct sequences of brief, standard therapy programs for all subjects that are followed by several months rest periods. Further, even subjects receiving full scale therapy can be included for comparative purposes. The important point is that longitudinal studies that include subjects exposed to therapy are still valuable when the therapy is carefully documented.

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REFERENCES

ADAMS, M. (1977). A clinical strategy for differentiating the normally nonfluent child and the incipient stutterer. *Journal of Fluency Disorders, 2,* 141–148.

ADAMS, M. (1986). *Stuttering, disfluency and fluency in children, a perspective overview.* Paper presented at the convention of the American Speech-Language-Hearing Association, Detroit. Abstract published in *Asha, 28,* 71.

ANDREWS, G. (1984). The epidemiology of stuttering. In D. Curlee & W. Perkins (Eds.), *Nature and treatment of stuttering.* San Diego: College-Hill Press.

ANDREWS, G., CRAIG, A., FEYER, A., HODDINOT, S., HOWIE, P., & NEILSON, M. (1983). Stuttering: A review of research findings and theories circa 1982. *Journal of Speech and Hearing Disorders, 48,* 226–246.

ANDREWS, G., & HARRIS, M. (1964). *The syndrome of stuttering.* London: Heinemann.

BARR, H. (1940). A quantitative study of specific phenomena observed in stuttering. *Journal of Speech Disorders, 5,* 277–280.

BECKER, K., WLASSOWA, N., ASATIANI, N., BELJAKOWA, L., & HEY, W. (1977). *Stottern.* Berlin: VEB Verlag Volk und Gesundheit. Cited in A. Preus (1981). *Identifying subgroups of stutterers.* Oslo, Norway: Universitetsforlaget.

BERLIN, A., (1954). *An exploratory attempt to isolate types of stuttering.* Unpublished doctoral dissertation. Northwestern University.

BLOOD, G., & SEIDER, R. (1981). The concomitant problems of young stutterers. *Journal of Speech and Hearing Disorders, 46,* 31–33.

BLOODSTEIN, O. (1961). The development of stuttering: III. Developmental stages. *Journal of Speech and Hearing Disorders, 26,* 67–82.

BLUEMEL, C. (1932). Primary and secondary stuttering. *Quarterly Journal of Speech, 18,* 178–200.

BRILL, A. (1921). Speech disturbances in nervous mental diseases. *Quarterly Journal of Speech, 9,* 129–135.

CANTER, G. (1971). Observations on neurogenic stuttering. A contribution to differential diagnosis. *British Journal of Communication Disorders, 6,* 139–143.

COMINGS, D., & COMINGS, B. (1987). A controlled study of Tourette Syndrome. I. Attention-deficit disorder, learning disorders, and school problems. *The American Journal of Human Genetics, 41,* 701–742.

COX, N., SEIDER, R., & KIDD, K. (1984). Some environmental factors and hypotheses for stuttering in families with several stutterers. *Journal of Speech and Hearing Research, 27,* 543–548.

DALY, D. (1981). Differentiating stuttering subgroups with Van Riper's developmental tracks: A preliminary study. *Journal of National Student Speech and Hearing Association, 9,* 89–101.

DAVIS, D. (1939). The relations of repetitions in the speech of young children to certain measures of language maturity and situational factors: Part I. *Journal of Speech and Hearing Disorders, 4,* 303–318.

DIXON, S. (1965). *Incipient stuttering symptoms and spontaneous remission of stuttered speech.* Paper presented at the convention of the American Speech-Language-Hearing Association, Chicago. Abstract published in *Asha, 10,* 371.

DIXON, S. (1971). Incipient stuttering and spontaneous remission of stuttered speech. *Journal of Communication Disorders, 4,* 99–110.

DOSTALOVA, N., & DOSUZKOV, T. (1966). Aspects of the three main categories of neurotic stuttering. *De Therapie Loquelis, 1,* 273–276.

DOUGLAS, E., & QUARRINGTON, B. (1952). The differentiation of interiorized and exteriorized secondary stuttering. *Journal of Speech and Hearing Disorders, 17,* 377–385.

EMERICK, L. (1966). *An evaluation of three psychological variables in tonic and clonic stutterers and in nonstutterers.*

FROESCHELS, E. (1921). Beitrage zur symtomatologie des stotterns. *Monatsschr Ohrenheilk, 55,* 1109–1112.

FROESCHELS, E. (1943). Pathology and therapy of stuttering. *Nervous Child, 2,* 148–161.

FRITZELL, B. (1974). The prognosis of stuttering in school-age children. *Proceedings of the 16th Congress of Logopedics and Phoniatrics,* Interlaken; Basel: Karger, 128–131.

GIOLAS, T., & WILLIAMS, D. (1958). Children's reactions to

nonfluencies in adult speech. *Journal of Speech and Hearing Research, 1,* 86–93.

GODAI, U., TATARELLI, R., & BONANI, G. (1976). Stuttering and tics in twins. *Acta Genetica Medicae et Gemellologiae, 25,* 369–375.

HINKLE, W. (1971). *A study of subgroups within the stuttering population.* Unpublished doctoral dissertation, Purdue University.

HUBBARD, C., & YAIRI, E. (1988). Clustering of disfluencies in the speech of stuttering and nonstuttering preschool children. *Journal of Speech and Hearing Research, 31,* 228–233.

INGHAM, R. (1976). "Onset, prevalence, and recovery from stuttering": A reassessment of findings from the Andrews and Harris study. *Journal of Speech and Hearing Disorders, 41,* 280–281.

JOHNSON, W., & ASSOCIATES (1959). *The onset of stuttering.* Minneapolis: University of Minnesota Press.

KIDD, K. (1984). Stuttering as a genetic disorder. In D. Curlee, & W. Perkins (Eds.), *Nature and treatment of stuttering.* San Diego: College-Hill Press.

KIDD, K. (1980). Genetic models of stuttering. *Journal of Fluency Disorders, 5,* 187–202.

KIDD, K., HEIMBUCH, R., & RECORDS, M. (1981). Vertical transmission of susceptibility to stuttering with sex-modified expression. *Proceedings of the National Academy of Science, 78,* 606–610.

KIDD, K., KIDD, J., & RECORDS, M. (1978). The possible cause of the sex ratio in stuttering and its implications. *Journal of Fluency Disorders, 3,* 13–23.

KIDD, K., HEIMBUCH, R., RECORDS, M., OEHLERT, G., & WEBSTER, R. (1980). Familial stuttering patterns are not related to one measure of severity. *Journal of Speech and Hearing Research, 23,* 539–545.

KROLL, A. (1976). *The differentiation of stutterers into interiorized and exteriorized groups.* Unpublished doctoral dissertation, Purdue University.

LUCHSINGER, R., & ARNOLD, G. (1965). *Voice-speech-language.* Belmont, California: Wadsworth Publishing Company.

LUDLOW, C., & COOPER, J. (1983). Genetic aspects of speech and language disorders: Current status and future directions. In C. Ludlow, & J. Cooper (Eds.), *Genetic aspects of speech and language disorders.* New York: Academic Press.

MILISEN, R., & JOHNSON, W. (1936). A comparative study of stutterers, former stutterers and normal speakers whose handedness has been changed. *Archives of Speech, 1,* 61–86.

NEWMAN, P. (1963). Adaptation performances of individual stutterers: Implications for research. *Journal of Speech and Hearing Research, 6,* 293–294.

OHASHI, Y. (1973). The development of stuttering in children. *Japanese Journal of Child Psychiatry, 14,* 142–166.

PINDZOLA, R., & WHITE, D. (1986). A protocol for differentiating the incipient stutterer. *Language, Speech, and Hearing Services In Schools, 17,* 2–15.

PREUS, A. (1981). *Identifying subgroups of stutterers.* Oslo, Norway: Universitetsforlaget.

PRINS, D., & LOHR, F. (1972). Behavioral dimension of stuttered speech. *Journal of Speech and Hearing Research, 15,* 61–71.

RYAN, B. (1984). *Stuttering in preschool children, a comparative longitudinal study.* Paper presented at the convention of the American Speech-Language-Hearing Association, San Francisco. Abstract published in *Asha, 26,* 102.

RILEY, G., & RILEY, J. (1982). Evaluating stuttering problems in children. *Journal of Childhood Communication Disorders, 6,* 15–25.

SCHWARTZ, H., & CONTURE E. (1988). Subgrouping young stutterers. *Journal of Speech and Hearing Research, 31,* 62–71.

SCHWARTZ, H., ZEBROWSKI, P., & CONTURE, E. (1986). *Behavior at the onset of stuttering.* Paper presented at the convention of the American Speech-Language-Hearing Association, Detroit. Abstract published in *Asha, 28,* 110.

SEIDER, R., GLADSTIEN, K., & KIDD, K. (1982). Language onset and concomitant speech and language problems in subgroups of stutterers and their siblings. *Journal of Speech and Hearing Research, 25,* 482–486.

SEIDER, R., GLADSTEIN, K., & KIDD, K. (1983). Recovery and persistence of stuttering among relatives of stutterers. *Journal of Speech and Hearing Disorders, 48,* 402–409.

SHEEHAN, J., & COSTLEY, M. (1977). A reexamination of the role of heredity in stuttering. *Journal of Speech and Hearing Disorders, 42,* 47–59.

SHEEHAN, J., & MARTYN, M. (1970). Stuttering and its disappearance. *Journal of Speech and Hearing Research, 13,* 279–289.

ST. ONGE, K. (1963). The stuttering syndrome. *Journal of Speech and Hearing Research, 6,* 279–289.

STROMSTA, C. (1965). A spectrographic study of disfluencies labeled as stuttering by parents. *De Therapia Vocis et Loquellae, 1,* 317–320.

VAN RIPER, C. (1971). *The nature of stuttering.* Englewood Cliffs: Prentice-Hall.

YAIRI, E. (1982). Longitudinal studies of disfluencies in two-year old children. *Journal of Speech and Hearing Research, 25,* 155–160.

YAIRI, E. (1983). The onset of stuttering in two- and three-year old children: a preliminary report. *Journal of Speech and Hearing Disorders, 48,* 171–177.

YAIRI, E., & AMBROSE, N. (1987). *The development and amelioration of early childhood stuttering.* Paper presented at the convention of the American Speech-Language-Hearing Association, New Orleans, Abstract published in *Asha, 29,* 71.

YAIRI, E., & LEWIS, B. (1984). Disfluencies at the onset of stuttering. *Journal of Speech and Hearing Research, 24,* 490–495.

YOUNG, M. (1975). Onset, prevalence, and recovery from stuttering. *Journal of Speech and Hearing Disorders, 40,* 49–58.

Chapter 7

SUBTYPING ADULT STUTTERERS FOR RESEARCH PURPOSES

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One way that the human mind extracts information from the world is by sorting things into separate mental groups and naming each group. This penchant for classifying everything has led us to subtype stuttering along many dimensions. Following is a list of some dimensions that come to mind; I have surely overlooked others. The list makes the point, however, that we can sort fluency and stuttering many differing ways. The question remains, however, which of these classification systems are useful to our understanding of stuttering and our ability to help stutterers become more fluent and which of these groupings are extraneous, perhaps blinding us to the discovery of some underlying core behavior that best describes stuttering.

DIMENSION	SUBTYPES
Severity	Mild, Moderate, Severe
Manifestation	Covert, Overt
Locus of Block	Labial, Laryngeal, Respiratory
Phonetic Features	Vowels, Consonants
Behaviors	Prolongations, Repetitions
Reactive Behaviors	Incipient, Treated, Nontreated
Etiology	Hysterical, Brain Damage, Cluttering, Stuttering, Normal Disfluency

CONTINUITY THEORY

Considering the last dimension first, there is very little confusion among people who present with hysterical speech interruptions due to stress such as combat fatigue, those who present with neurogenic/cortical 'stuttering', those who present cluttered speech, and those who present symptoms of stuttering. Bloodstein (1987, p. 85–87) differentiates these into groups according to presumed or known etiology. Clutterers, for example, do not exhibit the problem that stutterers do in initiating speech; their disfluencies are apt to be randomly spread throughout their utterances. Clutterers are not helped as much as stutterers by fluency enhancing conditions such as DAF or auditory masking.

There exists some confusion, however, between the disfluent speech of nonstutterers and that of stutterers. As Bloodstein writes further in his *Handbook on Stuttering:* "There are no stutter-type disfluencies that are not to be found in the speech of many nonstutterers." (p. 365). This overlap and confusion between normal disfluency and mild stuttering leads to an important theoretical question: Does fluency exist as a continuum from the most fluent nonstutterer to the most severe stutterer? Or, alternatively, is fluency a step function made up of TWO continua, the nonstuttering continuum of fluency differences and the stuttering continuum of fluency differences? The first dimension in the above table of subtypes (severity) would be played across the stuttered part of this step function. There is little research probing the differences between normal nonfluency and covert or mild stuttering. Thus, the continuity theory of fluency is a hypothesis waiting to be tested. There is some evidence that stutterers can tell the difference between real forced stuttering and fake stuttering (Bloodstein & Shogun, 1972) and that stuttering under DAF not only sounds different from real stuttering to listeners but feels different to stutterers (Neeley, 1961). The key difference seems to be a loss of control when the stuttering is real.

There are few studies of fluency that simultaneously investigate perception (by others) and production (by speakers). Studies that probe the perception of fluency according to listeners in parallel with neurophysiological probes of speech production in speakers would address the continuity issue. I would suggest that there is a continuum of fluency, but it is only perceptual. This continuous gradation of perceptual fluency obscures a discontinuity on some neurophysiological level in the speakers. In other words, according to some as yet unknown neurophysiological distinction, a speaker is either stuttering or not (a step function), despite the fact that there is perceptual overlap and confusion between covert or mild stuttering and the hesitant or otherwise disfluent speech of nonstutterers. My view of the perceptual continuity/neurophysiological discontinuity which exists among speakers is represented in Figure 1.

Are the speakers for which there is perceptual overlap (the mild stutterer and the normally disfluent speaker), producing their hesitations, repetitions, abnormally fast or slow speech, or whatever—differently from one another on some level, indicating production discontinuity? Or are they together on a speech production continuum that is as graded as the perceptual function? I would expect the neurophysiological level which splits the speakers into two functions to be quite central in the nervous system. The extent to which it would be reflected in more peripheral motor events is uncertain and a research question of strong theoretical importance.

DIFFERENTIAL DEFICIT THEORY

Considering the subtypes classified according to stuttering severity, research questions remain. Stuttering severity has historically been described according to behavioral criteria such as frequency and duration of stuttering episodes, occurrence of secondary symptoms such as signs of abnormal effort, and degree of speech naturalness.

Recently, there are published data from presumably fluent samples of the speech of stutterers that suggest certain physiological correlates to behaviorally determined degrees of severity. For example, Watson and Alfonso (1987) found differences in respiration patterns and respiratory-laryngeal organization that correspond to differences in stuttering severity. Specifically, *Mild* stutterers delay abdominal compression but use appropriate respiratory-laryngeal organization whereas *Severe* stutterers expand the respiratory system before opening the vocal folds and compress the system before the vocal folds are adducted. In another example, Borden, Baer, and Kenney (1985) found differences in vocal fold vibration patterns that also correlated with severity. Specifically, mild stutterers produced vocal fold vibration patterns indistinguishable from normal, whereas severe stutterers produced patterns with an unstable open phase and an abnormally rapid opening phase. In addition, voice onset was delayed for severe stutterers relative to that for nonstutterers and mild stutterers.

How should these and other such findings be interpreted? In the current literature there seem to be three interpretations of such findings:

1. There are chronic qualitative as well as quantitative differences in system deficits among severity subtypes (Watson & Alfonso, 1987)
2. There are no qualitative differences only quantitative differences so that for many mild stutterers the symptoms when fluent are too subtle to detect (Caruso, Conture, & Colton, 1988; Caruso, Gracco, & Abbs, 1987).
3. There are no chronic motor deficits, qualitative or quantitative, when stutterers are truly fluent; differences observed may be due to overlaid secondary behaviors, learned preventive strategies, or covert stuttering (Borden & Armson, 1988).

More research is needed to resolve the conflicting interpretations of these recent data. There is no doubt that some stutterers, even at their most fluent, speak more slowly than the average nonstutterer, but the fact that other stutterers speak as fast as anyone when truly fluent (Borden, 1983) forces the question of whether the slow rate is necessary to stuttering, a part of the core behavior, or some acquired adaptation. The other question is how fluent the perceptually 'fluent' samples used in such studies really are. Techniques to insure natural fluency such as adaptation and self-reporting when combined with multichannel physiological recordings might eventually yield consistent and distinctive profiles, if differences exist, for the fluent speech of mild, moderate, and severe stutterers. Similar studies might also yield perceptual and physiological profiles to distinguish normal disfluencies from mild stuttering as well as cluttering from stuttering.

Core Versus Reactive Behavior

The notion of some core behavior that constitutes stuttering versus reactive behaviors that are somewhat extraneous to the core, perhaps even disguising it, has most recently been advanced by Stromsta (1986). He views the core behavior to be a phonemic disruption; the speaker fails to proceed because he does not anticipate and prepare for the next sound as one would do in coarticulated coordinated speech. Thus, the vowel of the syllable is initiated but not completed. Prolongations and tonic blocks, seen by some as subtypes of stuttering behavior, are viewed by Stromsta as reactions to the basic phonemic disruption.

Another reactive progression of subtypes is suggested in the table at the beginning of this paper. The speaker reacts consciously to his previously unconscious incipient stuttering and further reacts with overlaid maladaptive behaviors or helpful strategies acquired during treatment.

In one sense the researcher might best approach core behavior by studying children, but in another sense one can argue, as indeed Stromsta does, that the core and reactive behaviors coexist in the adult stutterer and may be studied there. This suggests a critical area for research. At what level can one discover the events necessary and sufficient for stuttering to occur and what indices best describe it? When one peels away all the discovered and learned reactions to stuttering what is left? Is perhaps the core behavior, the stuttering itself, also a reaction? To what? To the expectation of stuttering? If so, what form does that expectation have? I think that the further we peel back the layers revealing the essence of the problem, the further up the nervous system we will go until we reach some cognitive/affective interaction with the neurophysiology of phonation. I am unable to suggest an effective research probe of this interaction with tech-

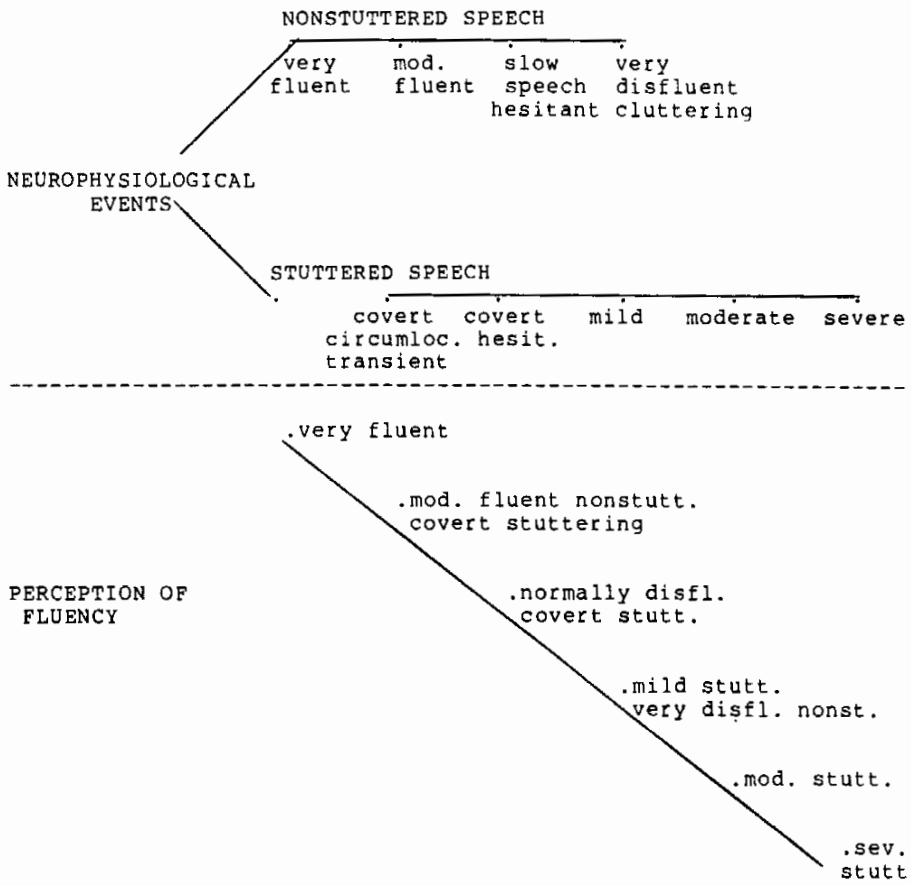

FIGURE 1. The perception of fluency seems to be a continuous function. On the other hand, the underlying motor events may in fact be discontinuous—that is, a speaker may be either stuttering or not stuttering.

niques currently available. Perhaps there is hope with blood flow studies. The Madison, Wisconsin group has suggested that it is a sequencing problem, indicting the Supplementary Motor Area on the superior cortex (Caruso, Abbs, & Gracco, 1988).

Anatomical Locus

Aside from differentiating cerebellar stuttering from possible SMA sequencing problems or some limbic involvement, there have been attempts to classify stutterers according to the dominant locus of the block at the more peripheral speech motor systems. For example, stutterers are described as *laryngeal* or *labial* stutterers. The larynx is clearly implicated (Conture, McCall, & Brewer, 1977; Freeman & Ushijima, 1978) but so, too, are supralaryngeal structures (Shapiro, 1980) and the respiratory system (Watson & Alfonso, 1987). A review of the extensive literature on laryngeal focus has been provided by Starkweather (1982). The argument over laryngeal focus continues, the debaters awaiting further evidence. Some argue that the problem lies not with a certain system but in the coordination of systems with one another (Perkins, Rudas, Johnson, & Bell, 1976; Stromsta, 1986; Van Riper, 1982), while others argue that coordination is not at fault when stutterers are truly fluent, rather it is a short circuit from another source to otherwise normal peripheral motor systems (Borden & Armson, 1988). My own bias on this issue is that the integrity of the motor systems has been demonstrated, but it is vulnerable to disruption from some other neural source, and the most vulnerable part of the system to such a disruption is the larynx because it requires vibration and delicate settings of resistance and positioning as well as ordinary movement. In our studies, we find lip/jaw with larynx coordination of movements for stutterers that is indistinguishable from nonstutterers, but voicing itself is often delayed or if started, aborted (Borden & Armson, 1988). This agrees with data that show laryngeal reaction times to be normal when recording EMG but slower than normal when recording the acoustic signal (Prosek, Montgomery, Walden, & Schwartz, 1979). Harrington (1987) finds that the schwa-sounding vowels of stuttered syllables are produced through appropriately shaped vocal tracts according to electropalatograms, while Howell, Williams, and

Vause (1987) report that the glottal source function during stuttering is lacking acoustic energy in the high frequency harmonics of its spectrum. These observations suggest a laryngeal focus to the stuttering which is often perceived by listeners as a failure to reach supralaryngeal (vocal tract) targets. Again, we see a lack of correspondence between what we perceive to be happening and actual neurophysiological events. It is the abnormal shortening of the stuttered vowel and loss of acoustic energy that causes it to be perceived as a neutralized vowel. The laryngeal focus hypothesis awaits further testing. It is confounded with the hidden core behavior problem, because there are so many overlaid behaviors in adult stutterers that even if there were a laryngeal focus to the core, the other systems are recruited to 'cope' with the problem and thus may camouflage the underlying and essential behavior. It may be that the best research approach in this case would be to study in detail a great many very mild stuttering events and attempt to find the common denominator among them that is distinct from perceptually similar normal nonfluencies.

There is another perspective from which some authors have suggested anatomical subtypes of stutterers: that of cerebral dominance. Because some but not all stutterers demonstrate a left ear advantage in listening to dichotic presentations of verbal stimuli (Brady & Berson, 1975; Rosenfeld & Goodglass, 1980), the idea of a subgroup of stutterers with a left hemisphere deficit has been suggested. This thinking likens stuttering to the "headache-multiple etiology" concept. Just as a headache can be a symptom of differing etiologies, it is tempting and facile to attribute stuttering to every theory that comes along and include them all in a multiple etiology explanation. As an example, let me quote from the medical dictionary on my desk (Taber, 1981): to stutter is to "hesitate and repeat or stumble spasmodically in speaking, due to a variety of causes, among them difficulty in pronouncing initial consonants caused by spasms of lingual and palatal muscles." (p. 1381) That settles everything; we needn't research further. Of course, there are hysterical speech interruptions and brain damage dysfluencies, but we can recognize them as different from stuttering.

Returning to cerebral dominance subtypes, Geschwind suggested that left cerebral hemisphere damage to the fetus from the male sex hormone testosterone might cause stuttering and would also account for the high incidence among males (Geschwind & Galaburda, 1985). Dichotic listening experiments may be probing an ability only distantly related to the problem of stuttering. After all, dichotic listening probes phonetic perception not production, and although it is expedient to harbor perception and production in a common cerebral hemisphere, even in normal individuals, this is not always the case (Milner, 1975).

UNIFIED THEORY OF STUTTERING

In my view, the most productive use of research time would be to seek the core behavior of stuttering across all subtypes. Subgroupings of stutterers and of disfluency types tell us more about overlaid coping behaviors than they do about the disorder itself. Of course, that means that it is prudent to divide speakers according to severity for data analysis and to divide disfluencies according to type in order to recognize the overlaid behaviors for what they are. If we find, as I have, that mild stutterers, when fluent, are indistinguishable for certain measures from nonstutterers but that severe stutterers when fluent differ from normal, we can infer from these results that the points of difference are due to reactive behaviors not to the stuttering itself. Also, subtypes of disfluencies such as repetitions, prolongations, hesitations may present an illusion of differences, whereas in reality the same sudden inability to make the next move may account for each behavior. Thus, movements and corresponding EMG signals may differ for the various manifestations of stuttering, but the immobility, the failure to proceed, although revealed in the movement and EMG records, may not be explained by them.

We must keep clear the difference between subtypes of fluency and subtypes of stuttering. Research efforts may be fruitful that explore subtypes of fluency (differences between normal disfluency and stuttering; differences between cluttering and stuttering). These studies should be designed to pair perceptual and physiological data. Research on stuttering, however, should be aimed at pinning down the factors necessary to stuttering, with subgroups of stutterers and disfluencies used to help distinguish primary and secondary factors.

REFERENCES

BLOODSTEIN, O. (1987). A handbook on stuttering. Chicago: National Easter Seal Society.

BLOODSTEIN, O., & SHOGUN, R. L. (1972). Some Clinical Notes on forced stuttering. *Journal of Speech and Hearing Disorders, 37*, 177–186.

BORDEN, G. J. (1983). Initiation versus execution time during manual and oral counting by stutterers. *Journal of Speech and Hearing Research, 26*, 389–396.

BORDEN, G. J., & ARMSON, J. (1988). *Coordination in stuttering: Order in chaos.* Unpublished manuscript.

BORDEN, G. J., BAER, T., & KENNEY, M. K. (1985). Onset of voicing in stuttered and fluent utterances. *Journal of Speech and Hearing Research, 28*, 363–372.

BRADY, J. P. & BERSON, J. (1975). Stuttering, dichotic listening, and cerebral dominance. *Archives of General Psychiatry, 32*, 1449–52.

CARUSO, A. J., ABBS, J. H., & GRACCO, V. L. (1988). Kinematic analysis of multiple movement coordination during speech in stutterers. *Brain, 111*, 439–455.

CARUSO, A. J., CONTURE, E. G., & COLTON, R. H. (1988). Selected temporal parameters of coordination associated with stuttering in children. *Journal of Fluency Disorders, 13*, 57–82.

CARUSO, A. J., GRACCO, V. L., & ABBS, J. H. (1987). A speech motor control perspective on stuttering: Preliminary observations. In H. F. M. Peters & W. Hulstijn (Eds.), *Speech motor dynamics in stuttering* (pp. 245–255). Wien and New York: Springer-Verlag.

CONTURE, E. G., MCCALL, G. N., & BREWER, D. W. (1977). Laryngeal behavior during stuttering. *Journal of Speech and Hearing Research, 20*, 661–668.

FREEMAN, F. J., & USHIJIMA, T. (1978). Laryngeal muscle activ-

ity during stuttering. *Journal of Speech and Hearing Research, 21,* 538–562.

GESCHWIND, N., & GALABURDA, A. M. (1985). Cerebral lateralization: Biological mechanisms, associations, and pathology: I. A hypothesis and a program for research. *Archives of Neurology, 42,* 429–459.

HARRINGTON, J. (1987). Coarticulation and stuttering: An acoustic and electropalatographic study. In H. F. M. Peters & W. Hulstijn (Eds.), 381–392.

HOWELL, P., WILLIAMS, M., & VAUSE, L. (1987). Acoustic analysis of repetitions in stutterers' speech. 371–380.

MILNER, B. (ED.). (1975). Hemisphere specialization and interaction. Cambridge, MA: M.I.T. Press.

NEELLEY, J. N. (1961). A study of the speech behavior of stutterers and nonstutterers under normal and delayed auditory feedback. *Journal of Speech and Hearing Disorders,* Monograph Supplement *7,* 63–82.

PERKINS, W. H., RUDAS, J., JOHNSON, L., & BELL, J. (1976). Stuttering: Discoordination of phonation with articulation and respiration. *Journal of Speech and Hearing Research, 19,* 509–522.

PROSEK, R. A., MONTGOMERY, A. A., WALDEN, B. E., & SCHWARTZ, D. M. (1979). Reaction time measures of stutterers and nonstutterers. *Journal of Fluency Disorders, 4,* 269–278.

ROSENFELD, D. B., & GOODGLASS, H. (1980). Dichotic testing of cerebral dominance in stuttering. *Brain and Language, 11,* 170–180.

SHAPIRO, A. (1980). An electromyographic analysis of the fluent and dysfluent utterances of several types of stutterers. *Journal of Fluency Disorders, 5,* 203–231.

STARKWEATHER, C W. (1982). Stuttering and laryngeal behavior: A review. ASHA Monograph No. 21.

STROMSTA, C. (1986). Elements of stuttering. Oshtemo, MI: Atsmorts.

TABER'S CYCLOPEDIC MEDICAL DICTIONARY. (1981). (14th edition). Philadelphia: F.A. Davis.

VAN RIPER, C. G. (1982). The nature of stuttering. Englewood Cliffs, NJ: Prentice-Hall.

WATSON, B., & ALFONSO, P. J. (1987). Physiological bases of acoustic LRT in nonstutterers, mild stutterers, and severe stutterers. *Journal of Speech and Hearing Research, 30,* 434–447.

E. PATHOPHYSIOLOGY

Chapter 8

NEUROMOTOR ASPECTS OF STUTTERING: LEVELS OF IMPAIRMENT AND DISABILITY

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Given how little is presently known about the neural processes which underly stuttering, it is worthwhile considering the perspective provided by the following comments of Lewis Thomas (1975):

> The great contemporary achievement of modern medicine is technology for controlling and preventing bacterial infection, but this did not fall into our laps with the appearance of penicillin and the sulfonamides. It had its beginnings in the final quarter of the last century, and decades of the most painstaking and demanding research were required before the etiology of pneumonia, scarlet fever, meningitis, and the rest could be worked out. Generations of energetic and imaginative investigators exhausted their whole lives on the problems. It overlooks a staggering amount of basic research to say that modern medicine began with the era of antibiotics.
>
> We have to face, in whatever discomfort, the real possibility that the level of insight into the mechanisms of today's unsolved diseases—schizophrenia, for instance, or cancer, or stroke, [or stuttering]—is comparable to the situation for infectious disease in 1875, with similarly crucial bits of information still unencountered (p. 136).

If one agrees that this general statement accurately reflects the state of affairs in the area of stuttering, then a stronger commitment to basic research is needed. In my opinion the issues to target for basic research on the problem of stuttering are those which can clarify how movement control in general, and speech movement in particular, are coded in the developing and mature nervous systems. This is not to say that the etiology of stuttering ultimately will be understood solely in terms of the motor systems of the brain (cf. Smith, Chapter 5), but there is now wide agreement that eventual understanding of the nature of stuttering will depend on a much greater knowledge of the neural basis of speech motor control (e.g., Caruso, Gracco, & Abbs, 1987; Neilson & Neilson, 1982; Starkweather, 1987; Wingate, 1983; Yeudal, 1985; Zimmermann, 1980c; Zimmermann, Smith, & Hanley, 1981). Some major issues in motor control are discussed here in order to provide a perspective on how related research may impact work on neuromotor aspects of stuttering. The reader is referred to two recent chapters for more in depth reviews of speech neurophysiology (Abbs & Welt, 1985; Barlow & Farley, 1989).

Issues in Motor Control

Motor control is generally seen as a goal-directed hierarchial process involving precise temporal patterns of neural activity in different brain centers such as the motor cortex, basal ganglia and cerebellum (Granit, 1980). In broad terms, the goal of research in motor control is to understand how various centers and pathways of the brain contribute to the regulation of muscle contraction during natural behaviors. For example, it has been suggested that the basal ganglia and supplementary motor area play a key role in initiating and regulating learned movements involving predictive type control, whereas the cerebellum and lateral premotor cortex are more involved in control requiring multisensory feedback where environmental conditions are less predictable (Goldberg, 1985).

A related issue in motor control concerns the nature of movement encoding in the CNS (Paillard, 1983). In other words, what parameters of movement (e.g., direction, velocity, relative timing) do different brain centers code and regulate prior to and during movement execution? This is likely to depend on both the nature of the movement and the neural pathways or centers being considered. For example, the basal ganglia and cerebellum may regulate distinct parameters of movement (see Massion, Paillard, Schultz, Wiesendanger, 1983; Towe & Luschei, 1981).

Research on motor control has focused on problems of limb movement in extrapersonal visual space, but in speech the crucial frame of reference is auditory and movements occur within the predictable spatial geometry of the respiratory airway. Thus, sound source, vocal-tract area and aerodynamic changes are probably coded at some levels of the CNS as intended targets for speech production. Achievement of such targets obviously involves the coordination of several distinct muscle groups,

and this is likely to require effective integration of somatosensory input.

An issue which has guided research in motor control for a number of years concerns how somatosensory input contributes to the control of muscle contraction. Research involving recording and stimulation of sensory neurons during movement has led to the view that voluntary movement involves selective gating or modulation of sensory input by corticofugal and cortico-cortical control processes. Evidence for such sensory gating processes in humans is provided by several recent studies in which somatosensory evoked potentials are recorded prior to and during movement (Cheron & Berstein, 1987; Cohen & Starr, 1987; Dietz, Berger, & Quintern, 1987; Tapia, Cohen, & Starr, 1987). Results of these studies indicate that there are systematic changes in the responsiveness of distinct regions of somatosensory and motor cortex to sensory input prior to and during different phases of movement. The role of this gating process is not well-understood, but it may facilitate much of the rapid, predictive, and automatic aspects of normal movement, particularly in the case of highly learned movements such as speech production.

An area of research that has strong implications for the issues of movement-parameter and sensory coding concerns how muscles and movements are represented in the motor cortex. Work in this area is particularly important, because the motor cortex represents the major point of neural integration just prior to the alpha motoneuron pools. It has long been known that surface electrical stimulation of area 4 will produce discrete movements in different body parts depending on the site of stimulation (see Abbs & Welt, 1985; Humphrey, 1986). In recent years, techniques of cortical microstimulation and recording have revealed several important features of motor cortex organization (Asanuma, Zarzecki, Jankowska, Hongo, & Marcus, 1979; Georgopoulos, Kettner, & Schwartz, 1988; Huang, Hiraba, & Sessle, 1989; Kwan, MacKay, Murphy, & Wong, 1978). For example, (a) Individual muscles tend to be represented within discrete columns of neurons in area 4. (b) A given muscle is represented at multiple noncontiguous sites within areas 4 and 6. (c) Area 4 neurons are most responsive to somatosensory input from the regions where they produce muscle activation. (d) Area 6 plays a key role in controlling the pattern of activity of area 4 neurons and in regulation of associated somatosensory input. (e) Area 4 neurons are strongly modulated by changes in movement direction with individual neurons showing their highest firing rates for unique directions of movement.

Future research on these aspects of motor cortex organization is likely to have a growing influence on how EMG data are interpreted relative to CNS control of speech movements and speech dysfluency. For example, Moore, Smith, and Ringel (1988) found that antagonist jaw muscles tend to co-contract during speech but show reciprocal patterns of activation during mastication and nonspeech periodic movement of the jaw. Thus, antagonist muscles may show differing degrees of reciprocity or coactivation depending on the requirements of a particular task. Abbs and Welt (1985) suggest that multiple representation of individual muscles in motor cortex may underly such flexibility in muscle activation patterns, as well as the tendency for some speakers (e.g., stutterers) to have more difficulty producing certain speech sounds.

Stuttering Impairment, Disability, and Handicap

In trying to review and suggest directions for research on the neuromotor aspects of stuttering, I have found it helpful to refer to a general model of human disorder which is now widely applied in various health professions. The essential feature of this model is the distinction made between the concepts of impairment, disability, and handicap. As defined by the World Health Organization (1980) and elaborated by others (Frey, 1984), *impairment* refers to physiological, anatomical, or psychological loss, abnormality, or injury. *Disability* refers to restriction or lack of ability to perform a human activity, and handicap to inability or lack of opportunity to perform a socially expected role. I have used the concepts of impairment and disability here because (a) they may be easily related to this widely used model of human disorder, (b) they provide a logical manner for categorizing a wide range of relevant studies, and (c) they suggest productive ways of organizing research efforts on the neuromotor aspects of stuttering.

In the present context, *impairment* refers to disorders of the motor systems underlying speech production which are causally related to but are not identified as speech dysfluency. Here I use the term *motor systems* to include the centers of the brain known to be involved in regulation of motoneuron output (i.e., primary motor cortex, premotor cortex, basal ganglia, cerebellum, and primary somatosensory cortex). Converging pathways from areas which mediate emotional response and which are likely to be involved in the intentional aspects of movement control (e.g., anterior cingulate cortex) may also be thought of as part of the motor system. A further distinction can be made between structural and functional impairment. These two concepts are elaborated below.

The concept of *stuttering disability* refers to the range of perceptual and physical events associated with speech dysfluency, including the actions of somatic systems not directly involved in speech production. As I use the term, speech dysfluency requires a perceptual component, that is, for speech to be dysfluent it must be identified as such by a listener.

Stuttering handicap refers to the adverse effects of stuttering disability on the individual's capacity for normal speech communication, and all this implies in terms of personal growth, educational experience, occupational choice and social function. Issues of stuttering handicap are beyond the scope of the present paper and will not be considered here, but one could effectively argue that a greater independent research effort is needed in this area. Such work certainly could have considerable benefit for individual stutterers.

Structural Impairment in Stuttering

Structural impairment refers to anatomical abnormalities which are causally related to speech dysfluency. It is assumed that such abnormalities are associated with the nervous system, for example, differences in the density or structure of synaptic terminals as projected from one brain center to another, or differences in brain chemistry as reflected in the distribution of neurotransmitters across brain centers involved in speech production.

To my knowledge, there are presently no direct data on the nature of structural impairment associated with developmental stuttering. However, the validity of the concept is suggested by the numerous cases of stuttering acquired in association with neurologic lesion. Such cases indicate that some structural and/or chemical change due to acute trauma, degenerative disease, or toxic response to chemical agents has caused the stuttering. The conventional wisdom is that acquired and developmental stuttering are vastly different disorders. This view, however, is based chiefly on subjective description of the peripheral aspects of speech production, and evidence has yet to be presented that the central neural mechanisms of dysfluency are not similar in the two cases. At our present level of understanding of brain function and stuttering, I think it is prudent not to make strong assumptions in this regard.

A striking aspect of the reported cases of stuttering acquired in adults is the diversity of associated neurologic conditions; for example, strokes affecting primarily the cerebral cortex (Helm, Butler, & Canter, 1980; Rosenbek, Messert, Collins, & Wertz, 1978), penetrating lesions which affect primarily the basal ganglia (Ludlow, Rosenberg, Salazar, Grafman, & Smutok, 1987), and phenytoin toxicity which may have adverse effects on the cerebellum (McClean & McLean, 1985). This general observation emphasizes the complexity of the problem, because the common symptom of speech dysfluency can apparently be associated with neural dysfunction at a number of distinct sites within the brain. The diversity of neuropathologic conditions associated with acquired stuttering is not surprising given that the motor control process depends on interdependent function of several anatomically distributed systems (Goldberg, 1985; Massion, Paillard, Schultz, & Wiesendanger, 1983). For example, the basal ganglia and cerebellum are likely to perform interdependent functions in neural coding for speech movements as their output is integrated within the premotor and primary motor cortices. Thus, the neural basis of stuttering may be associated with anomalies in any one of a number of constituent brain centers which comprise the neural circuitry underlying speech movement control. This idea is consistent with the view expressed by Pauls (Chapter 4) that stuttering is likely to be "etiologically *and* genetically heterogeneous."

Given that developmental stuttering typically occurs in the absence of other adverse neurologic conditions, how can we study relevant structural impairment directly in humans? First, it should be feasible to undertake the type of postmortem anatomical studies which have been carried out on individuals with dyslexia (Galaburda, Sherman, & Rosen, 1985). Just as cases of acquired dyslexia provide important information for the study of developmental dyslexia (Geschwind, 1985), acquired stuttering may provide important clues for where and how to look at structural impairment in cases of developmental stuttering. Cases of acquired stuttering suggest that studies of the gross structural and archetectonic characteristics of stutterers' brains should not be restricted to cerebral cortex but should also address possible differences in the basal ganglia, cerebellum, thalamus, and brain stem. A growing literature indicates that deviation from the normal pattern of brain asymmetry associated with speech processing may be an important causal factor in stuttering (see reviews by Moore, Chapter 9 and Yeudall, 1985, and this may have some structural basis).

Various brain imaging techniques such as computerized X-ray tomography (CT scan), cerebral blood flow, positron emission tomography (PET scan), magnetic resonance imaging (MRI), and electroencephalography (EEG) can provide additional approaches to the study of structural impairment in stuttering. Data acquired with these methods may be more or less amenable to structural interpretation depending on the particular technique and the evolving understanding of what constitutes evidence for a structural difference within the nervous system. In this regard, it now appears that relatively subtle changes may occur in the morphology of neurons in association with their repeated use, disuse, and exposure to toxic elements (Milgram, MacLeod, & Petit, 1987).

EMG and EEG evoked potential data are amenable to structural interpretation, because responses are limited to a restricted set of sensory or sensorimotor pathways. These methods entail controlled stimulation of a particular sensory system and signal averaging of associated EMG or EEG events. By utilizing relevant data on basic neuroanatomy and physiology, and evoked potential studies in human surgical patients, reasonable structural interpretations have been given to evoked potential data in humans (e.g., Jenner & Stephens, 1982; Leandri, Parodi, Zattoni, & Favale, 1987; McClean & Smith, 1982). This approach has been taken in studies of the auditory system of stutterers (e.g., Hall & Jerger, 1978), and modern techniques involving chronic recording of neuronal activity in unanesthetized awake animals are providing a more refined basis for a similar application of trigeminal evoked potentials. For example, recent studies involving intracortical microstimulation and recording in nonhuman primates are leading to a much improved map of how orofacial somatosensory systems project to the motor cortex (Huang, Hiraba, & Sessle, 1989).

Published studies have shown little or no difference between stutterers and nonstutterers somatic reflexes during nonspeech tasks requiring voluntary maintenance of constant levels of muscle activity (McClean, 1987, 1989); Neilson, Andrews, Guitar, & Quinn, 1979; Smith & Luschei, 1983). These negative findings may be due in part to experimental methods which are not sufficiently refined to reveal what are likely to be subtle differences

in the sensorimotor systems of stutterers and nonstutterers. For example, parametric analyses relating the effects of stimulus magnitude and background EMG may be required to effectively assess possible differences in sensorimotor pathways in stutterers and nonstutterers (Tatton, Beddingham, Verrier, & Blair, 1984).

Functional Impairment in Stuttering

Functional impairment refers here to abnormalities in speech motor system physiology which are not identified as speech dysfluency. Such abnormality may be reflected in subtle differences in the physical aspects of speech production, for example, increased voice onset times or deviant timing of structural movement; or it may involve abnormal aspects of speech motor system function during nonspeech performance such as in tracking or reaction-time tasks. Work in this area is based in part on the assumption that abnormalities in motor system output during fluent speech reflect anomalies in the neuromotor system which are causally related to dysfluent speech.

The physical characteristics of perceptually fluent speech of stutterers have been studied by a number of investigators using EMG, kinematic, and acoustic measures (e.g., Adams, 1987; Alphonso, Watson, & Baer, 1987; Borden, Baer, & Kenney, 1985; Caruso et al., 1987; Caruso, Abbs, & Gracco, 1988; Conture, Colton, & Gleason, 1988; Goldsmith, 1984; Guitar, Guitar, Neilson, O'Dwyer, & Andrews, 1988; Healey & Gutkin, 1984; Hillman & Gilbert, 1977; Prosek, Montgomery, Walden, & Hawkins, 1987; Zimmermann, 1980a). In general, the findings are mixed with respect to possible differences in the fluent speech of stutterers and nonstutterers, and the magnitudes of physical differences tend to be small. This latter observation is not surprising, because the acceptable ranges of acoustic parameters required for perceptually fluent speech are also most likely small. This emphasizes, of course, the importance of maximizing the degree of resolution obtained with the measurement tools and controlling for extraneous subject variables such as sex and size which may contribute to physical differences in speech output.

Analysis of speech structure movements holds particular promise for understanding the neural basis of stuttering, because events at the kinematic level may reflect important aspects of neural coding in motor control (cf. Gracco & Abbs, 1988; Paillard, 1983). Also, simultaneous measures of different structures provide a direct basis for assessing coordination. This is particularly relevant, because initial studies indicate that physical differences in stutterers' fluent speech are most evident in measures of the relative timing of different structures (e.g., voice onset time or timing of lip and jaw movement).

Zimmermann (1980a) carried out a cineradiographic study of lip, jaw, and tongue movements of the fluent productions of simple syllables in adult stutterer and nonstutterers. As a group the stutterers showed longer movement durations, reduced movement displacements and velocities, and greater asynchrony of lip and jaw timing. Speakers were not matched, however, in terms of sex or oral-facial size, two variables which are likely to contribute to interspeaker differences in parameters of speech structure movement. In a subsequent study of lip and jaw motion, these variables were controlled (Goldsmith, 1984), and no statistically significant differences between stutterers and nonstutterers were obtained.

Caruso, Abbs, and Gracco (1988) described several aspects of lip-jaw kinematics in the fluent speech of stutterers and nonstutterers. Generally they saw few differences in the two groups, but they did note marked abnormalities in the relative timing of lip and jaw velocity peaks in the stutterers. In nonstutterers velocity peaks during lip closure consistently followed the temporal sequence upper lip, lower lip, and jaw, with interstructure time lags of approximately 5–20 ms. Among the stutterers this sequence was never the predominant temporal pattern, and they showed greater intrasubject variability across multiple repetitions of the same utterance. Caruso and his collaborators speculate that these timing anomalies are related to dysfunction of CNS control processes involving the supplementary motor cortex.

Functional impairment associated with stuttering may also be observed in tests of nonspeech motor performance involving tracking or reaction-time paradigms (e.g., Bakker & Brutten, 1987; Neilson & Neilson, 1982; Nudelman, Herbrich, Hoyt, & Rosenfield, 1987; Sussman & MacNeilage, 1975). The use of tracking paradigms holds particular promise, because one may vary the nature of tracking tasks in such a way as to assess differential impairment of distinct motor centers or systems. For example, Flowers (1978) has noted that Parkinson subjects track unpredictable visual targets at normal error levels, but show marked deficits for predictable targets where the predictive capacities of the basal ganglia are more likely to be required.

Stuttering Disability

Stuttering disability refers to perceptually dysfluent speech and associated physical events such as static posturing and tremor. The problem of understanding the neural basis of the physical aspects of dysfluency is similar to that associated with a variety of neuromotor disorders. That is, some behaviors may represent negative signs which are the direct result of the neurologic disorder or structural impairment, some may be positive signs which are secondary or release phenomena, and others may be compensatory signs reflecting adaptations or coping strategies on the part of the individual. A taxonomy of abnormal movements and postures associated with stuttering might eventually be organized around such distinctions. However, it is probably not practical to develop such a taxonomy until we have a better picture of the peripheral physiologic events associated with stuttering at different age levels.

Descriptive studies of stuttering have employed EMG, kinematic, acoustic, and aerodynamic measures, (e.g., Fibiger, 1971; Howell & Vause, 1986; Hutchinson, 1974;

Zimmermann, 1980b). Individual studies have tended to focus on relatively distinct issues, with varying degrees of basic versus clinical emphasis. For example, Fibiger (1971) used EMG to study facial muscle activity relative to the issue of physiological tremor and the potential role of the cerebellum in stuttering. Hutchinson (1974), on the other hand, employed aerodynamic measures to describe unique aspects of individual stutterer's behavior and to suggest the utility of such information for stuttering therapy.

The analysis of EMG signals from different muscles during moments of stuttering has the potential for providing important clues on the nature of its central neural mechanisms. Initial EMG studies focused on the issue of cerebral dominance (see review by Moore, 1984). These and subsequent studies (Freeman & Ushijima, 1978; Fibiger, 1971; Shapiro, 1980) suggest that moments of stuttering are characterized by increased levels of muscle activation, co-contraction of antagonist muscles, and tremor in the 6–12 Hz range.

McClean, Goldsmith, and Cerf (1984) addressed these various observations in an analysis of lip-muscle EMG during the fluent and dysfluent speech of stutterers. Contrary to earlier reports, they observed reciprocal activation of antagonist lip muscles during fluent and dysfluent speech, and reduced levels of lower-lip depressor muscle during sustained bilabial blocks. Thus, speech dysfluency was not characterized simply by a gross increase in the activation levels of all muscles. The fact that reciprocal activation of antagonist muscles was retained during dysfluencies suggests that the disrupted input was not to motoneurons per se but to higher level neural circuitry involved in automatic regulation of the timing of antagonist muscle contraction.

Smith (1989) has recently compared the muscle activity associated with the dysfluent speech of 10 stutterers with the fluent speech of 10 nonstutterers. Consistent with the findings of Moore et al. (1988), she has observed extensive co-contraction of anatagonist jaw muscles in the fluent speech of nonstutterers. In the stutterers she observed equivalent levels of co-contraction to the nonstutterers fluent speech and reduced EMG levels in some muscles. Smith also presents evidence that during dysfluent speech individual stutterers tend to show idiosyncratic patterns of aberrant muscle activation. This observation has important implications for how we view the nature of central motor processes underlying dysfluency, and it certainly warrants additional study.

Roadblocks and Future Directions

Major barriers to understanding the neural basis of stuttering include our lack of knowledge of the anatomy and physiology of neural systems underlying normal speech production and insufficient technology for transducing the neural processes underlying speech. As would be expected from this, much of the related research on stuttering has not been guided by narrowly defined hypotheses on the neural mechanisms of speech motor control. Instead, hypotheses have been of a general nature or the primary goal has been to describe the peripheral aspects of stutterers' fluent and dysfluent speech. Careful description of the peripheral aspects of stuttering are certainly important, but where possible physiologic studies in this area need to be framed in terms of the likely neural systems and mechanisms underlying motor control by the CNS.

In our present attempts to understand the nature of neural mechanisms underlying speech dysfluency, it is logical to focus on those aspects of disordered movement which already have a strong interpretive base in the motor control literature. For example, the analysis of stuttering tremor provides a viable target for more analytic studies of speech dysfluency (Borden, Baer, & Kenney, 1985; Fibiger, 1971; McFarland, Smith, Moore, & Weber, 1986; McClean et al., 1984; Smith, 1989). A considerable amount is known about the physiology of tremor and its magnitude may be readily determined from amplitude spectra on EMG or kinematic signals (Freund, 1983; Hunker & Abbs, 1984; Stein & Lee, 1981). Tremor in speech muscle systems may be particularly relevant to stuttering because of its similarity at a peripheral level to speech sound repetitions.

Previous kinematic studies of stutterers' fluent speech have involved relatively simple statistical methods and small numbers of subjects. Future studies might effectively apply multivariate statistics (Kleinbaum & Kupper, 1978) to analysis of several kinematic variables in larger groups of subjects. For example, we have recently had some success in discriminating stutterers from nonstutterers and predicting stuttering severity levels from a limited set of speech kinematic measures (McClean, Kroll, & Loftus, 1989). Factor analysis might also be usefully applied to partially assess the nature of primary control parameters in speech motor output (Keller, 1987).

Another important area for future study concerns the patterns of muscle contraction seen during speech dysfluency. As our understanding of cortical motor output is enhanced, we may be able to make reasonable interpretations of the range of muscle activation patterns associated with fluent and dysfluent speech. Research to date suggests that degree of co-contraction and level of muscle activation *within* muscle systems (e.g., lips or tongue) do not reflect the principal features of disordered input to motoneurons during dysfluency. This conclusion is consistent with the view that the critical problem in stuttering relates to temporal coordination of multiple structures or muscle systems (Kent, 1984). This of course emphasizes the need for more studies in which the activity of several muscle systems are transduced during moments of stuttering (e.g., Caruso, Conture, & Colton, 1988; Smith, 1989; Zimmermann, 1980b).

Analysis of cortical potentials which occur prior to muscle contraction for speech is presently feasible, and such potentials probably reflect important aspects of the organization of ensuing movements (Wohlert & Larson, 1988; Zimmermann & Knott, 1974). This is a promising line of research, because anomalies in premovement cortical potentials are very likely to be associated with

speech dysfluencies. Future EEG analysis of prespeech cortical events in stutterers might logically focus on activity in cortical areas 5, 6, and 7 which are implicated in anticipation and preparatory processes preceding normal movement (Deecke & Kornhuber, 1978; Goldberg, 1985; Libet, 1985; MacKay & Crammond, 1987; Okano & Tanji, 1987; Roland, Larsen, Lassen, & Skinhoj, 1980; Tanji & Kurata, 1985).

A technique which has considerable promise for the study of sensory processing in speech production involves unpredictable mechanical loading of peripheral structures and analysis of associated compensatory adjustments (e.g., Abbs & Gracco, 1984; Gracco & Abbs, 1988). Preliminary use of these methods has been made with stutterers (Caruso, Abbs, & Gracco, 1988). It may be practical in the future to combine load-perturbation techniques with modern approaches to EEG analysis (e.g., Crammond, 1988) to evaluate how different regions of the sensorimotor cortex are gated or modulated for speech movement control. Unfortunately cortical potentials recorded during speech are likely to be contaminated by cranial muscle potentials (Leandri et al., 1987). This is a major technical problem to be overcome, but one which warrants a concerted effort.

Clinical descriptions of stuttering behavior typically involve perceptual classification of the frequency of different types of speech dysfluency. It is now practical to supplement these perceptual descriptions with physiologic and acoustic measures. The relative value of such data for clinical practice will depend on our ability to provide valid neurophysiologic interpretations of the abnormal movements associated with speech dysfluency. This will require analytic and correlational studies which relate variables at different levels of stuttering impairment and disability.

REFERENCES

ABBS, J., & GRACCO, V. (1984). Control of complex motor gestures: Orofacial muscle responses to load perturbations of the lip during speech. *Journal of Neurophysiology, 52,* 705–723.

ABBS, J., & WELT, C. (1985). Structure and function of the lateral precentral cortex: Significance for speech motor control. In R. Daniloff (Ed.), *Speech science: Recent advances.* San Diego: College-Hill Press.

ADAMS, M. (1987). Voice onset times and segment durations of normal speakers and beginning stutterers. *Journal of Fluency Disorders, 12,* 133–139.

ALPHONSO, P., WATSON, C., & BAER, T. (1987). Measuring stutterers' dynamical vocal tract characteristics by X-ray microbeam pallet tracking. In H. Peters & W. Hulstijn (Eds.), *Speech motor dynamics in stuttering,* New York: Springer-Verlag.

ASANUMA, H., ZARZECKI, P., JANKOWSKA, E., HONGO, T., & MARCUS, S. (1979). Projection of individual pyramidal tract neurons to lumbar motor nuclei of the monkey. *Experimental Brain Research, 34,* 73–89.

BAKKER, K., & BRUTTEN, G. (1987). Labial and laryngeal reaction times of stutterers and nonstutterers. In H. Peters & W. Hulstijn (Eds.), *Speech motor dynamics in stuttering.* New York: Springer-Verlag.

BARLOW, S., & FARLEY, G. (1989). Neurophysiology of speech. In D. Kuehn, M. Lemme, & J. Baumgartner (Eds.), *Neurobiology of speech, language and hearing.* Boston: Little, Brown.

BORDEN, G., BAER, T., & KENNEY, M. (1985). Onset of voicing in stuttered and fluent utterances. *Journal of Speech and Hearing Research, 28,* 363–372.

CARUSO, A., GRACCO, V., & ABBS, J. (1987). A speech motor control perspective on stuttering: preliminary observations. In H. Peters & W. Hulstijn (Eds.), *Speech motor dynamics in stuttering.* New York: Springer Press.

CARUSO, A., ABBS, J., & GRACCO, V. (1988). Kinematic analysis of speech multiple movement coordination in stutterers. *Brain, 111,* 439–455.

CARUSO, A., CONTURE, E., & COLTON, R. (1988). Selected temporal parameters of coordination associated with stuttering in children. *Journal of Fluency Disorders, 13,* 57–82.

CHERON, G., & BORENSTEIN, S. (1987). Specific gating of the early somatosensory evoked potentials during active movement. *Electroencephalography and Clinical Neurophysiology, 67,* 537–548.

COHEN, L., & STARR, A. (1987). Localization, timing and specificity of gating of somatosensory evoked potentials during active movement in man. *Brain, 110,* 451–467.

CONTURE, E., COLTON, R., & GLEASON, J. (1988). Selected temporal aspects of coordination during fluent speech of young stutterers. *Journal of Speech and Hearing Research, 31,* 640–653.

CRAMMOND, D. (1988). *The utilization by cortex of sensory information for the production of movement: A neurophysiological study in human subjects and macaque monkeys.* Doctoral dissertation, University of Toronto.

DEECKE, L., & KORNHUMBER, H. (1978). An electrical sign of participation of the mesial "supplementary" motor cortex in human voluntary finger movement. *Brain Research, 159,* 473–476.

DIETZ, V., BERGER, W., & QUINTERN, J. (1987). Task-dependent gating of somatosensory transmission in two different motor tasks in man: falling and writing. *Neuroscience Letters, 75,* 288–292.

FIBIGER, S. (1971). Stuttering explained as a physiological tremor. *Speech Transmission Laboratory—Progress Report,* (2–3, 1–23). Stockholm: Royal Institute of Technology.

FLOWERS, K. (1978). Some frequency response characteristics of parkinsonism on pursuit tracking. *Brain, 101,* 19–34.

FREEMAN, F., & USHIJIMA, T. (1978). Laryngeal muscle activity during stuttering. *Journal of Speech and Hearing Research, 21,* 538–562.

FREUND, H. (1983). Motor unit and muscle activity in voluntary motor control. *Physiological Reviews,* 63, 387–436.

FREY, W. (1984). Functional assessment in the '80s. In A. Halpern & M. Fuhrer (Eds.), *Functional assessment in rehabilitation,* (pp. 11–43). Baltimore: Paul H. Brookes.

GALABURDA, A., SHERMAN, G., & ROSEN, G. (1985). Developmental dyslexia: Four consecutive cases with cortical anomalies. *Annals of Neurology, 18,* 222–233.

GEORGOPOULOS, A., KETTNER, R., & SCHWARTZ, A. (1988). Primate motor cortex and free arm movements to visual targets in three-dimensional space. II. Coding of the direction of movement by a neuronal population. *Journal of Neuroscience, 8,* 2928–2937.

GESCHWIND, N. (1985). The biology of dyslexia. In D. Gray & J. Kavanagh (Eds.), *Biobehavioral measures of dyslexia.* Parkton, MD: York Press.

GOLDBERG, G. (1985). Supplementary motor area structure and function: review and hypothesis. *The Behavioral and Brain Sciences, 8,* 567–616.

GOLDSMITH, H. (1984). *Articulatory kinematics in stuttering and nonstuttering adults.* Unpublished doctoral dissertation, University of Washington.

GRACCO, V., & ABBS, J. (1988). Central patterning of speech movements. *Experimental Brain Research, 71,* 515–526.

GRANIT, R. (1980). *The purposive brain.* Cambridge, MA: MIT Press.

GUITAR, B., GUITAR, C., NEILSON, P., O'DWYER, N., & ANDREWS, G. (1988). Onset sequencing of selected lip muscles in stutterers and nonstutterers. *Journal of Speech and Hearing*

Research, 31, 28–35.

HALL, J., & JERGER, J. (1978). Central auditory function in stutterers. *Journal of Speech and Hearing Research, 21,* 324–337.

HEALEY, E., & GUTKIN, B. (1984). Analysis of stutterers' voice onset times and fundamental frequency contours during fluency. *Journal of Speech and Hearing Research, 27,* 219–225.

HELM, N., BUTLER, R., & CANTER, G. (1980). Neurogenic acquired stuttering. *Journal of Fluency Disorder, 5,* 269–279.

HILLMAN, R., & GILBERT, H. (1977). Voice onset time for voiceless stop consonants in the fluent reading of stutterers and nonstutterers. *Journal of the Acoustical Society of America, 61,* 610–611.

HOWELL, P., & VAUSE, L. (1986). Acoustic analysis and perception of vowels in stuttered speech. *Journal of the Acoustical Society of America, 79,* 1571–1579.

HUANG, S., HIRABA, H., & SESSLE, B. (1989). Input-output relationships of primary face motor cortex in the monkey (Macaca fascicularis). *Journal of Neurophysiology, 61,* 350–362.

HUMPHREY, D. (1986). Representation of movements and muscles within the primate precentral motor cortex: historical and current perspectives. *Federation Proceedings, 45,* 2687–2699.

HUNKER, C., & ABBS, J. (1984). Physiological analyses of parkinsonian tremors in the orofacial system. In M. McNeil, J. Rosenbek, & A. Aronson (Eds.), *The dysarthrias: Physiology, acoustics, perception, management.* San Diego: College-Hill Press.

HUTCHINSON, J. (1974). Aerodynamic patterns of stuttered speech. In L. Webster & L. Furst (Eds.), *Vocal tract dynamics and dysfluency.* New York: Speech and Hearing Institute.

JENNER, J., & STEPHENS, J. (1982). Cutaneous reflex responses and their central nervous pathways studied in man. *Journal of Physiology, 333,* 405–419.

KELLER, E. (1987). Factors underlying tongue articulation in speech. *Journal of Speech and Hearing Research, 30,* 223–229.

KENT, R. (1984). Stuttering as a temporal programming disorder. In R. Curlee & W. Perkins (Eds.), *Nature and treatment of stuttering: New directions.* San Diego: College-Hill Press.

KLEINBAUM, D., & KUPPER, L. (1978). *Applied regression analysis and other multivariate methods.* Boston, MA: Duxbury Press.

KWAN, H., MACKAY, W., MURPHY, J., & WONG, Y. (1978). Spatial organization of precentral cortex in awake primates. II. Motor outputs. *Journal of Neurophysiology, 41,* 1120–1131.

LEANDRI, M., PARODI, C., ZATTONI, J., & FAVALE, E. (1987). Subcortical and cortical responses following infraorbital nerve stimulation in man. *Electroencephalography and Clinical Neurophysiology, 66,* 253–262.

LIBET, B. (1985). Unconscious cerebral initiative and the role of conscious will in voluntary action. *The Behavioral and Brain Sciences, 8,* 529–566.

LUDLOW, C., ROSENBERG, J., SALAZAR, A., GRAFMAN, J., & SMUTOK, M. (1987). Site of penetrating brain lesions causing chronic acquired stuttering. *Annals of Neurology, 22,* 60–66.

MACKAY, W., & CRAMMOND, D. (1987). Neuronal correlates in posterior parietal lobe of the expectation of events. *Behavioral and Brain Research, 24,* 167–179.

MASSION, J., PAILLARD, J., SCHULTZ, W., & WIESENDANGER, M. (1983). *Neural coding of motor performance.* Berlin: Springer-Verlag.

MCCLEAN, M. (1987). Surface EMG recording of the perioral reflexes: Preliminary observations on stutterers and nonstutterers. *Journal of Speech and Hearing Research, 30,* 283–287.

MCCLEAN, M. (1989). Gain and spatial characteristics of human lip-muscle reflexes. *Brain Research 503,* 16–21.

MCCLEAN, M., & SMITH, A. (1982). The reflex response of single motor units in human lip muscles to mechanical stimulation. *Brain Research, 251,* 65–75.

MCCLEAN, M., GOLDSMITH, H., & CERF, A. (1984). Lower-lip EMG and displacement during bilabial disfluencies in adult stutterers. *Journal of Speech and Hearing Research, 27,* 342–349.

MCCLEAN, M., & MCLEAN, A. (1985). A case report of stuttering acquired in association with phenytoin use for post-head injury seizures. *Journal of Fluency Disorders, 10,* 241–255.

MCCLEAN, M., KROLL, R., & LOFTUS, N. (1989). Discriminant analysis of lip-jaw motion in stutterers and nonstutterers. *Asha, 31,* 66.

MCFARLAND, D., SMITH, A., MOORE, C., & WEBER, C. (1986). Relationship between amplitude of tremor and reflex responses of the human jaw-closing system. *Brain Research, 366,* 272–278.

MILGRAM, N., MACLEOD, C., & PETIT, T. (1987). *Neuroplasticity, learning, and memory,* New York: Allan Liss.

MOORE, W. (1984). Central nervous system characteristics of stutterers. In R. Curlee & W. Perkins (Eds.), *Nature and treatment of stuttering: New directions.* San Diego: College-Hill Press.

MOORE, C., SMITH, A., & RINGEL, R. (1988). Task-specific organization of activity in human jaw muscles. *Journal of Speech and Hearing Research, 31,* 670–680.

NEILSON, M., & NEILSON, P. (1982). Speech motor control and stuttering. In D. Russel & B. Abernathy (Eds.), *Proceedings of the Otago Symposium on Motor Memory and Control* (pp. 69–80). Otago, New Zealand: University of New Zealand Press.

NEILSON, P., ANDREWS, G., GUITAR, B., & QUINN, P. (1979). Tonic stretch reflexes in lip, tongue and jaw muscles. *Brain Research, 178,* 311–328.

NUDELMAN, H., HERBRICH, K., HOYT, B., & ROSENFIELD, D. (1987). Dynamic characteristics of vocal frequency tracking in stutterers and nonstutterers. In H. Peters & W. Hulstijn (Eds.), *Speech motor dynamics in stuttering,* New York: Springer-Verlag.

OKANO, K., & TANJI, J. (1987). Neuronal activities in the primate motor fields of the agranular frontal cortex preceding visually triggered and self-paced movement. *Experimental Brain Research, 66,* 155–166.

PAILLARD, J. (1983). Introductory lecture: The functional labelling of neural codes. In J. Massion, J. Paillard, W. Schultz, & M. Wiesendanger (Eds.), *Neural coding of motor performance* (pp 1–19). Berlin: Springer-Verlag.

PROSEK, R., MONTGOMERY, A., WALDEN, B., & HAWKINS, D. (1987). Formant frequencies of stuttered and fluent vowels. *Journal of Speech and Hearing Research, 30,* 301–305.

ROSENBEK, J., MESSERT, B., COLLINS, M., & WERTZ, R. (1978). Stuttering following brain damage. *Brain and Language, 6,* 82–96.

ROLAND, P., LARSEN, B., LASSEN, N., & SKINHOJ, E. (1980). Supplementary motor area and other cortical areas in organization of voluntary movements in man. *Journal of Neurophysiology, 43,* 118–136.

SHAPIRO, A. (1980). An electromyographic analysis of the fluent and dysfluent utterances of several types of stutterers. *Journal of Fluency Disorders, 5,* 203–231.

SMITH, A. (1989). Neural drive to muscles in stuttering. *Journal of Speech and Hearing Research, 32,* 252–264.

SMITH, A., & LUSCHEI, E. (1983). Assessment of oral-motor reflexes in stutterers and normal speakers: Preliminary observations. *Journal of Speech and Hearing Research, 26,* 322–328.

STARKWEATHER, W. (1987). Laryngeal and articulatory behavior in stuttering: Past and future. In H. Peters & W. Hulstijn (Eds.), *Speech motor dynamics and stuttering.* Springer-Verlag: New York.

STEIN, R., & LEE, R. (1981). Tremor and clonus. In V. Brooks (Ed.), *Handbook of physiology (Vol. 2).* Bethesda, MD: American Physiological Society.

SUSSMAN, H., & MACNEILAGE, P. (1975). Hemispheric specialization for speech production and perception in stutterers. *Neuropsychologia, 13,* 19–26.

TANJI, J., & KURATA, K. (1985). Contrasting neuronal activity in supplementary and precentral motor cortex of monkeys. I. Responses to instructions determining motor responses to forthcoming signals of different modalities. *Journal of Neurophysiology, 53,* 129–141.

TAPIA, M., COHEN, L., & STARR, A. (1987). Selectivity of attenuation (i.e., gating) of somatosensory potentials during voluntary movement in humans. *Electroencephalography and Clinical Neurophysiology, 68,* 226–230.

TATTON, W., BEDDINGHAM, W., VERRIER, M., & BLAIR, R. (1984). Characteristic alterations in the response to imposed wrist displacement in Parkinsonian rigidity and dystonia musculorum deformans. *The Canadian Journal of Neurological Sciences, 11,* 281–287.

THOMAS, L. (1975). *The lives of a cell: Notes of a biology watcher.* Bantam Books: Toronto.

TOWE, A., & LUSCHEI, E. (1981). *Handbook of behavioral neurobiology: Motor coordination. Volume 5.* New York: Plenum Press.

WINGATE, M. (1983). Speaking unassisted: Comments on a paper by Andrews et al. *Journal of Speech and Hearing Disorders, 48,* 255–263.

WOHLERT, A., & LARSON, C. (1988). Premovement cerebral motor potentials in oral and laryngeal tasks. *Asha, 30,* 177.

WORLD HEALTH ORGANIZATION (1980). *International classification of impairments, disabilities, and handicaps: A manual of classification relating to the consequences of disease.* New York: World Rehabilitation Fund.

YEUDALL, L. (1985). A neuropsychological theory of stuttering. *Seminars in Speech and Language, 6,* 197–223.

ZIMMERMANN, G. (1980a). Articulatory dynamics of fluent utterances of stutterers and nonstutterers. *Journal of Speech and Hearing Research, 23,* 95–107.

ZIMMERMANN, G. (1980b). Articulatory behaviors associated with stuttering: A cineradiographic analysis. *Journal of Speech and Hearing Research, 23,* 108–121.

ZIMMERMANN, G. (1980c). Stuttering: A disorder of movement. *Journal of Speech and Hearing Research, 23,* 122–136.

ZIMMERMANN, G., & KNOTT, J. (1974). Slow potentials of the brain related to speech processing in normal speakers and stutterers. *Electroencephalography and Clinical Neurophysiology, 37,* 599–607.

ZIMMERMANN, G., SMITH, A., & HANLEY, J. (1981). Stuttering: In need of a unifying concept. *Journal of Speech and Hearing Research, 46,* 25–31.

Chapter 9

PATHOPHYSIOLOGY OF STUTTERING: CEREBRAL ACTIVATION DIFFERENCES IN STUTTERERS VS. NONSTUTTERERS

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When one considers the Central Nervous System's (CNS) possible relationship to stuttered verbal behavior several fundamental questions come to mind which include: Are there hemispheric asymmetry differences between stutterers and nonstutterers for information processing? How do these differences effect fluency? Do the differences represent functional or morphological differences between stutterers and nonstutterers? Although none of these questions can be fully answered from the available research, the first has been the most thoroughly addressed, and has been the focus of the majority of hemispheric asymmetry studies conducted with stutterers. Much of the available research has sought to discover interhemispheric differences between stutterers and nonstutterers and to relate these findings to different patterns of hemispheric activation between groups. Indeed, little information has been presented which addresses the other two questions. This focus on interhemispheric difference may reflect, in part, procedural limitations, conceptual limitations, or a combination of the two. In this paper a review of the research on hemispheric processing in stutterers will be presented. A discussion of the needs and directions for future research in this area will also be presented.

A few disclaimers, by way of clarification, are needed at the start of this paper. First, and perhaps most important for many readers, I have no formal "theory" about hemispheric processing as related to stutterers and stuttering. The reason for this stems from my inductive approach to theory construction which requires that a theory emerge from the available data. From my perspective, we do not have sufficient data to construct a theory at this time. At best we can develop hypotheses about the relationship between hemispheric processing and stuttering; but we must be prepared to make modifications in these hypotheses as new data emerge from the laboratory. Another concern is the use of laboratory contrived tasks and conditions in dichotic and tachistoscopic methods that do not necessarily reflect what one does in life. These data may negatively influence theory construction. Indeed, blood flow and spontaneous EEG procedures have been more successful in representing the activation of both hemispheres in hemispheric processing paradigms, yet fewer of these data are available to help theory construction.

During the last two decades we have seen a major resurgence of interest in the neurophysiological components of stuttering. With modern investigative techniques, researchers have explored possible differences between stutterers and normal speakers on various dimensions of central nervous system function. Although the existence of such differences was first suggested by Orton (1927) and Travis (1931), technological advances have encouraged modern researchers to study CNS function and dysfunction in ways that were not available to those innovative pioneers. (q.v., Moore & Boberg, 1987; for a descriptive discussion of the methodologies for the study of hemispheric processing.)

Evidence of differences in hemispheric asymmetries between stutterers and nonstutterers has been found using a variety of techniques. Greater right hemispheric activation has been reported for meaningful linguistic stimuli with dichotic listening (Curry & Gregory, 1969; Davenport, 1979; Perrin & Eisenson, 1970; Quinn, 1972; Sommers, Brady, & Moore, 1975), tachistoscopic viewing (Hand & Haynes, 1983; Moore, 1976; Plakosh, 1978), hemispheric alpha asymmetries (Boberg et al., 1983; Moore & Haynes, 1980; Moore & Lang, 1977; Moore & Lorendo, 1980; Moore, 1987; Ray & Moore, 1988; Wells & Moore, 1988), average evoked responses (Ponsford, Brown, Marsh, & Travis, 1975; Zimmerman & Knott, 1974), cortical blood flow (Wood et al., 1980), bimanual hand tasks (Fitzgerald, Cooke, & Greiner, 1984; Webster, 1988), and sequential finger tapping (Webster, 1985, 1986).

When reviewing studies that have investigated hemispheric processing in stutterers, it is important to compare the stimulus, task, and subject variables that have or have not been controlled. As with research with normal subjects (q.v., Friedman & Polson, 1981), it is not unusual to find different results in investigations that have used dissimilar procedures (e.g., high imagery words vs. low

imagery words; recall tasks vs. recognition tasks; uncontrolled levels of stuttering severity vs. controlled levels of severity; mixed gender vs. controlled gender; children vs. adults; nonsense syllables vs. meaningful linguistic stimuli). For example, dichotic investigations which have used nonsense syllables typically have reported no differences between stutterers and nonstutterers, or have reported a greater proportion of stutterers with a left ear preference score (Brady & Berson, 1975; Strong, 1978; Rosenfield & Goodglass, 1980; Liebetrau & Daly, 1981; Cimorell-Strong et al., 1983; Blood, 1985). On the other hand, investigations that used meaningful linguistic stimuli (words) have reported consistent right hemispheric involvement in stutterers under dichotic listening procedures (Curry & Gregory, 1969; Perrin & Eisenson, 1970; Quinn, 1972; Sommers, Brady, & Moore, 1975). Stuttering severity must also be considered in hemispheric processing studies. Moore (1986) reported a relationship between stuttering severity and hemispheric alpha suppression. His findings revealed a decrease in right hemisphere alpha suppression as stuttering severity increased. Differences have also been reported between stuttering children and adults (q.v., Bloodstein, 1987). The apparent inconsistencies between studies must be evaluated relative to the procedural differences amongst studies.

Left hemispheric processing of nonsense syllables, which are heavily dependent on phonetic discrimination, is a highly predictable event given that the left auditory cortex is primarily specialized for the processing of brief transient sounds and the retention of sound patterns (Thatcher, 1980). What is not predictable is the observation that larger proportions of stutterers process phonetic information right hemispherically under dichotic listening procedures. This observation might suggest that many more stutterers than nonstutterers process phonetic information with different processing strategies. Or in the extreme case, it may reflect differences in structural asymmetries in the stutterer's brain. In fact, a recent investigation by Strum, Black, and Naeser (1987) reported that CT scan measurements showed atypical asymmetries in the occipital brain regions in stutterers. Importantly, atypical occipital asymmetries have been associated with equal or increased right planum temporale length (Pieniadz & Naeser, 1984). The recent observation of a longer and wider right occipital region in stutterers provides preliminary evidence of structural differences in the brains of stutterers which may relate to their use of different processing strategies compared to nonstutterers.

A pervasive CNS mechanism has been suggested in studies with stutterers using visual tachistoscopic procedures with meaningful linguistic stimuli. The observation of a left visual field preference in stutterers has been replicated by several independent investigators (Moore, 1976; Plakosh, 1978; Hand & Haynes, 1983; Wilkins, Webster, & Morgan, 1984). It appears that language, whether written or spoken, is processed in the right hemispheres of many more stutterers than nonstutterers. These findings emphasize the need for a language processing explanation of stuttering that includes written as well as spoken language.

Webster (1985, 1986) using the behavioral repetitive dual task sequential finger tapping task, interpreted his data as being consistent with a neuropsychological model of stuttering that includes an element of increased left-hemisphere vulnerability to interference by concurrent right-hemisphere activity. In a more recent study using the behavioral bimanual writing task Webster (1988) suggested that the overall pattern of results implicated the supplementary motor area in the mediation of stuttering, possibly through relatively ungated callosal pathways (an interpretation similar to that of Caruso, Abbs, & Gracco, 1988).

Although behavioral input-output methods (dichotic, tachistoscopic, and finger tapping) have been popular in investigations with stutterers they have several limitations which limit one's view of brain-language relationships. A primary limitation is not knowing what happens to the lateralized input stimulus once it is being processed by the brain. Generalizations to language processing have been made in many dichotic studies that have used CV nonsense syllables. There is little evidence that lateralization of input infers lateralization of information processing (Valsiner, 1983). Also to infer that hemispheric processing of CV nonsense syllables is similar to that of meaningful linguistic stimuli (words and phrases) is not correct.

Electrophysiological paradigms afford researchers an opportunity to observe electrophysiological activity in the hemispheres simultaneously during information processing. Importantly, these procedures allow the investigator to study aspects of dynamic information processing/hemispheric activation over time. Two electrophysiological procedures have been used with stutterers to investigate hemispheric asymmetries: Averaged evoked responses and hemispheric alpha asymmetries.

Averaged evoked responses (AER) have been used by Ponsford, Brown, Marsh, and Travis (1975) to investigate hemispheric differences between stutterers and nonstutterers. Potentials were evoked with meaningful words embedded in phrases. Results indicated that normals' responses were accompanied by greater left hemisphere AER changes. Stutterers showed a reversal of this trend, with greater changes in the right hemisphere. Shenker, Seitz, and Doehring (1980) used AER procedures with stutterers and nonstutterers during a single fluent word reading task. Their results revealed that AER amplitudes for the left hemisphere were significantly larger than those of the right hemisphere for normal speakers, but there was no significant left hemisphere advantage for either severe or mild stutterers. They interpreted their findings as support for a hypothesis of a lower margin of cerebral dominance in stutterers during fluent speaking conditions.

Zimmerman and Knott (1974) used the contingent negative variation (CNV) method of AER with stutterers and nonstutterers during verbal and nonverbal tasks. Slow potential shifts were recorded from Cz, iF'3 and iF'4. Vertex CNVs were similar in normal speakers and stutterers in the verbal and nonverbal tasks. However, differences between groups were found at the lateral elec-

trodes in the verbal tasks. Preceding speech, four of five of the normal speakers showed a larger shift in the left hemisphere than in the right, while 88% of the stutterers showed a right greater than left asymmetry. Similar relationships were found in the verbal condition in which the subjects did not speak.

Pinsky and McAdam (1980) also used the CNV method with stutterers. In their study nonlinguistic stimuli were used under two conditions. One condition required subjects to press a button with each thumb simultaneously when a tone stopped. For the second condition subjects uttered the same previously identified fluent word at the termination of a tone over several trials. In contrast, Zimmerman and Knott's study required stutterers to utter each word presented whether fluent or not. Pinsky and McAdam concluded that their results provided insufficient evidence to support hemispheric asymmetry differences between stutterers and nonstutterers. An explanation for the differences between the Zimmerman and Knott and Pinsky and McAdam results may reside in the procedures used. In fact, a CNV study completed by Prescott and Andrews (1984) reported a greater late CNV over the right hemisphere prior to speech responses in both stutterers and nonstutterers. They appropriately concluded that attention needs to be paid to the nature of the subject's response in CNV research with stutterers and nonstutterers.

Differences in alpha suppression of stutterers and nonstutterers have been reported in several research projects conducted by Moore and his co-workers (Moore, 1984; Moore, 1986; Moore & Haynes, 1980; Moore & Lang, 1977; Moore, Craven, & Faber, 1982; Moore & Lorendo, 1980; Ray & Moore, 1988; Wells & Moore, 1988). These studies used single words of high and low imagery, sentences of different syntactical form, connected reading passages, tones, a quasi-conversational task, and words of differing arousal value. Memory conditions of recall and/or recognition, and listening, formulation and production tasks have been used. Greater right hemispheric alpha suppression in posterior electrodes (but not frontal electrodes) for stuttering subjects has been consistently observed across studies. Alpha suppression in nonstuttering subjects has varied, as would be expected, relative to stimulus and task demands. Indeed, nonstuttering female subjects have shown the greatest variations in alpha suppression with manipulation of stimulus, task, and electrode placement variables. Moore (1986) has also reported a significant correlation between stuttering severity and right hemispheric alpha suppression.

Results obtained by Boberg, Yeudall, Schopflocher, and Bo-Lassen (1983) agreed in general with those of Moore and his co-workers. They reported greater alpha suppression in the right posterior frontal areas of their stutterers. However, after three weeks of therapy, during which stuttering was reduced by training in the use of a slow speech rate and gentle onsets of phonation, the stutterers demonstrated left hemisphere alpha suppression during the performance of verbal tasks. The observation of greater left alpha suppression with increased fluency has also been reported by Moore (1984) using an EMG biofeedback procedure for treatment. Wood et al. (1980) have reported increased right frontal blood flow during stuttering and increased left frontal flows with decreased stuttering. Pinsky and McAdam reported no difference in alpha suppression between stutterers and nonstutterers after treating their data categorically. However, alpha power ratios were computed by taking the left hemisphere/right hemisphere value for each of 5 left hemisphere tasks and 4 right hemisphere tasks and then calculating the mean for each subject on each type of task. Due to artifact the inferior frontal recordings were eliminated, and only the temporal-parietal leads were considered. Their method of categorical inspection of collapsed means for the various tasks may have obscured important differences between tasks and groups.

According to the results from two recent hemispheric alpha asymmetry studies (Ray & Moore, 1988; Wells & Moore, 1988) stutterers and nonstutterers do not differ in hemispheric alpha asymmetries during noninformational resting tasks. Differences in asymmetries that do exist have been observed only during active processing of meaningful linguistic stimuli. The observation of no hemispheric asymmetries for the stuttering and nonstuttering groups during the noninformational resting tasks provides a baseline from which to measure asymmetric effects during linguistic tasks between groups.

One of the motivating factors in conducting hemispheric asymmetry research with stutterers has been the desire to find evidence to support or refute the Orton-Travis hypothesis of a lack of cerebral "dominance" for speech in stutterers. Approaching hemispheric activation from an hemispheric processing point of view provides a different interpretation than when it is approached from the standpoint of cerebral laterality. Current research suggests that "hemispheric dominance" or "lateralization" should be viewed in relation to the information processing strategies available to the left and right hemispheres, and not simply in terms of the nature of the stimuli to be processed (verbal or nonverbal). The nature of right hemispheric processing is qualitatively distinct from left hemispheric processing. The right hemisphere has been characterized as a holistic, parallel, nonsegmental, and time-independent processor contrasted with the left, which has been seen as analytic, linear, sequential, and time-dependent (Gordon, 1979; Moscovitch, 1977). The left hemisphere is uniquely suited for the processing, discrimination, and retention of brief transient sounds and temporal patterns (Thatcher, 1980). The right hemisphere is more involved in the processing and storage of continuous information, longer transients, and nonverbal pattern sequences. Resources from the right and/or the left hemispheres may be involved in language processing, depending on stimulus dimensions, task requirements, and the subject's approach to the task. Suggestions that hemispheric processing research with stutterers is "fraught with inconsistencies" (Bloodstein, 1987) may more correctly reflect the differences in stimulus, task, and subject variables amongst the studies reported and not inconsistencies in the findings *per se*. Apparent inconsistency gives way to consistency when

stimulus, task, and subject variables are considered against the background of hemispheric processing research conducted with normal subjects.

Perhaps the one consistent finding that has been shown is that many more stutterers than nonstutterers demonstrate right hemispheric processing for *meaningful* linguistic stimuli. This has been observed across a variety of hemispheric processing methods including behavioral dichotic and tachistoscopic input-output procedures, blood flow procedures, and electrophysiological procedures. The observation of no hemispheric alpha asymmetry differences between stutterers and nonstutterers during noninformational resting tasks provides a baseline from which to measure asymmetric effects during informational linguistic tasks between groups.

There are far fewer available data that allow us to address the question of how these differences affect fluency. Yet, three independent investigations have reported a shift from right to left hemispheric processing with increases in fluency (Boberg et al., 1983; Moore, 1984; Wood et al., 1980) and one investigation (Moore, 1986) has reported a significant correlation between right hemispheric alpha suppression and stuttering severity. In the studies by Boberg et al. (1983) and Wood et al. (1980) the right hemisphere has been implicated in motor programming deficits in stutterers with the observation of reduced alpha and increased blood flow in the anterior right hemisphere during periods of increased stuttering.

Stuss and Benson's (1984) review of the functions of the frontal lobes may provide a partial rationale for the differences seen in stutterers. These authors reviewed studies which showed impairment of verbal fluency and voluntarily controlled ordering of actions in left frontal lobe damaged patients. These findings can be used to support a motor programming disruption hypothesis of stuttering (Kent, 1983; Moore & Haynes, 1980) linked to the use of right hemispheric strategies/resources for motoric programming or the interference of normal left hemispheric mechanisms by the right hemisphere (q.v., Webster, 1988). Sussman and MacNeilage (1975) used an auditory tracking task requiring subjects to match the pitch of a tone heard in one ear to the varying pitch of a tone heard in the other. They concluded that stutterers have a less distinct lateralization of speech-related auditory sensorimotor integration. Caruso, Abbs, and Gracco (1988) recently reported that stutterers do not manifest general problems of coordination of speech movement. However, they did find that stuttering appears to be associated with a specific impairment in multiple movement coordination associated with sequencing of speech movements. It has been hypothesized that sequencing of movement is one function of the supplementary motor area (SMA) (Goldberg, 1985; Roland, Larsen, Lassen, & Skinho, 1980). Caruso et al. (1988) speculated that the SMA may be involved in stuttering disorders. Boberg et al. (1983) and Wood et al. (1980) findings provide some support for this speculation; however, their observations are restricted to Broca's area and not the SMA.

Investigations conducted by Ray and Moore and Wells and Moore have not supported greater right frontal alpha suppression in stutterers. These investigators have reported no significant differences between stutterers and nonstutterers for anterior frontal areas (Broca's Area) during listening and production tasks. Differences were observed only for the posterior temporo-parietal areas. No studies have investigated alpha asymmetries in the area of the SMA, but this appears to be a potentially important area of research. Speculations about the involvement of the SMA are provocative and should lead to empirical investigations to study its involvement in stuttering.

Investigations which have addressed the functional and/or pathophysiological nature of hemispheric asymmetries in stutterers are scarce. A recent investigation reported by Strub, Black, and Naeser (1987) studied atypical cortical asymmetries with CT scans and CV dichotic procedures in two left-handed siblings with developmental stuttering. Results revealed a longer and wider right occipital region in one subject and an atypical equal occipital length and width in the other subject. Neither subject was found to have a significant dichotic ear preference score. Strub et al. (1987) suggested that the atypical occipital asymmetries may be associated with equal or increased right planum temporale length (q.v., Pieniadz & Naeser, 1984). These findings were interpreted as supporting the theory that stuttering may be related to anomalous cerebral dominance, both on functional as well as structural bases.

Whether or not results form hemispheric asymmetry studies using behavioral or electrophysiological procedures reflect structural differences in the brains of stutterers is difficult to address. Clearly, the research in hemispheric processing with normals has indicated that there are both exogenous and endogenous components involved in hemispheric activation (q.v., Donchin & McCarthy, 1979) The exogenous components consist of the characteristics of the stimuli while the endogenous components involve task demands and individual subject processing styles which affect hemispheric activation. Perceived task demands will greatly influence the manner in which verbal and nonverbal stimuli are processed. For example, Willis, Wheatley, and Mitchell (1979) have shown that spatial stimuli, under analytic task demands, is processed left hemispherically, while Gates and Bradshaw (1977) found that trained musicians process music more left hemispherically than nonmusicians. A dichhaptic stimulation procedure was used by Webster and Thurber (1977) to determine if hand differences could be influenced by the use of differential problem-solving strategies. Half of their subjects were instructed to approach the haptic learning and recognition tasks in a "sequential" manner, and half to approach them in a "holistic" manner. Results revealed that the degree of left hand superiority on the tasks was influenced by the manner in which the subjects were instructed to approach the dichhaptic tasks. Using a double reversal single-subject design Jenkins and Moore (1985) studied the effects of visual feedback on hemispheric alpha asymmetries and processing strategies reported by a male subject during a linguistic task. Results indicated that the subject was able to identify specific behavioral strategies that

were associated with differential left or right hemispheric alpha suppression. Thus, during a linguistic task, the subject was able to exhibit control of right and left hemispheric processing strategies sufficient to produce changes in alpha hemispheric asymmetries.

Friedman and Polson (1981) have developed a framework for understanding how cerebral specialization of function contributes to the flexibility of human information processing. They suggest that the difficulties involved in obtaining reliable, replicable data in hemispheric asymmetry research may reflect the fact that tasks can usually be performed with a number of different resource compositions, each of which draws in varying amounts from the two hemispheres. According to Friedman and Polson: "Tasks that allow an assortment of resource compositions will tend to be easily influenced by parameter manipulations and may thus produce the most variable results across different populations of subjects . . . Thus, in addition to the fact that tasks vary in the extent to which they demand resources from one or the other hemisphere, we assume that subjects vary in the extent to which the resources of either hemisphere can be efficiently applied to performance." (p. 1055)

Perhaps the strongest argument in favor of functional asymmetries in stutterers comes from the observation that measures of hemispheric activation have been found to change following behavioral intervention to reduce stuttering. Boberg et al. (1983) reported that after three weeks of therapy in the use of a slow rate and gentle onsets of phonation that stuttering was reduced and the stutterers showed left posterior frontal alpha suppression compared to right alpha suppression prior to the behavioral treatment. Moore (1986) has also reported shifts from right alpha suppression to left suppression with increases in fluency which accompanied an EMG biofeedback procedure to reduce stuttering. These findings suggest that behavioral intervention, which modified the way stutterers performed on speaking tasks, was importantly involved in manipulating hemispheric alpha asymmetries and, inferentially, hemispheric processing strategies. Compliance with task demands (increased verbal fluency) may have required the stutterers to draw primarily from the resource compositions of the left hemisphere. This argues in favor of functional mechanisms being associated with anomalous hemispheric asymmetries in stutterers.

It is apparent that far too few investigations have been designed to address the question of functional or morphological asymmetries between stutterers and nonstutterers. Moore and Haynes (1980) suggested that stutterers may be more dependent upon the use of a nonsegmental processor (the right hemisphere) in dealing with segmental information at a linguistic-motor planning level. Indeed, one of the important functions of the left hemisphere is its specialization toward discrimination and memory for brief temporal patterns of auditory input. Structurally, the planum temporale of the left hemisphere is typically larger than that on the right and is importantly involved in the processing of brief temporal transitions (Thatcher, 1980) which are typical of the transition formats for consonants. The left hemisphere is more efficient in processing brief transient sounds and the storage of verbal pattern sequences. In contrast, the right hemisphere is more efficient in the processing and storage of continuous or steady-state information and nonverbal pattern sequences (q.v., Thatcher, 1980). Stuss and Benson (1984) in a review of the functions of the frontal lobes noted that temporal ordering of events is hemisphere specific, with verbal fluency and voluntarily controlled ordering of actions being left hemispheric functions.

A hypothetical concern with stuttering is the speed of linguistic-motoric processing and its effects on fluency. The above data indicate that the right hemisphere has a longer processing base compared to the left and that it is less important in verbal fluency and ordering of actions. Thus, the predominant use of the right hemisphere for linguistic-motor planning tasks may lead to, among other things, disruptions in motor speech fluency and speed of processing. The observation that stutterers generally become more fluent when using prolonged syllable timed speech may reflect the right hemisphere's capability for processing longer durations/transitions. Too, the linguistic loci of stuttering (q.v., Moore & Boberg, 1987) appear to be associated with language variables which the right hemisphere is less capable of processing and supports an hypothesis of increased dependency on right hemispheric nonsegmental processing being associated with stuttering. Whether the use of right hemispheric processing strategies is due to morphological or functional causes is a matter for future research. Yet, it seems apparent that models of stuttering and hemispheric processing must relate the different processing capabilities of the left and right hemispheres to behavioral observations of stuttering. The observation that slow, prolonged speech patterns increase "artificial" fluency in stutterers fits nicely into a right hemispheric processing model of stuttering considering the temporal parameters of left and right hemispheric processing and voluntary controlled ordering of actions.

ROADBLOCKS AND FUTURE DIRECTIONS

The above review of the hemispheric processing research with stutterers make it obvious that there are many unanswered questions. However, it is equally obvious that there exist several fruitful directions that can be taken which may lead us to the answers to many of these questions and provide data for the formulation of a CNS model of stuttering.

Perhaps one of the most basic roadblocks in this area of research is the need for a revision of our conceptualization of hemispheric processing. Given the current state of knowledge it is no longer tenable to view the left hemisphere simply as a "verbal" processor and the right hemisphere as a "nonverbal" processor. Indeed, the brain is a far more complex system than a two-compartment black box (Valsiner, 1983). A conceptualization of

the left and right hemispheres as mutually independent, but with complimentary resources functioning in conjunction with subcortical structures for information processing would alter the direction and interpretation of research in hemispheric processing with stutterers. Perhaps one of the greatest roadblocks to the development to a more contemporary neurophysiological conceptualization of brain and language is the lack of courses in the neurosciences in both our undergraduate and graduate curriculums. One of our future directions should be the inclusion of coursework in the neurosciences and the recognition that such coursework will help in understanding the process of which normal and disordered language is the outcome. Unfortunately, much of the hemispheric processing literature is contained in journals and other publications outside of speech-language pathology. An obvious paucity of hemispheric processing investigations are found in the speech-language pathology literature.

There has been general agreement that many more stutterers than nonstutterers demonstrate greater right hemispheric processing for meaningful linguistic stimuli. Yet, where the processing takes place in the right hemisphere remains controversial. Electrophysiological studies by Moore and co-workers have reported hemispheric alpha asymmetry differences over the right temporo-parietal area during language processing but not over the right frontal areas. Boberg and his associates have reported greater right hemispheric asymmetries over Broca's area but not the temporal or parietal regions. Recent investigations (Caruso et al., 1988; Webster, 1988) have suggested that the supplementary motor area, which has been hypothesized to be involved with sequencing of movement, may be involved in the disorder of stuttering. Unfortunately, the many behavioral input-output studies (e.g., dichotic studies) conducted with stutterers have provided little data that address questions of localization of processing and activation except in the most general terms. As mentioned above, the temporal processing characteristics of the left and right hemispheres may be important in understanding stuttered verbal behavior and for understanding the methods used to increase both "artificial" and "natural" fluency.

An important need in this area is to determine where in the right hemisphere, and under what stimulus and task conditions, anomalous processing is found in stutterers. We need to know if right hemispheric processing is associated with general anomalous processing for linguistic and motor functions or if it is restricted to certain areas of the right hemisphere (i.e., temporo-parietal, Broca's, and/or supplementary motor area) or to particular processing functions (i.e., motor sequencing). Answers to these questions may help to explain the language, memory and motor speech differences sometimes seen in stutterers (q.v., Moore, 1984; Moore & Boberg, 1987).

We have very little data which address processing in stutterers over time. For example, we have no data which shows the participation of the left and right hemispheres during listening or speaking tasks across time. If changes in relative hemispheric participation could be revealed over a processing epoch and correlated with episodes of fluent and dysfluent speech we might better understand the variability seen in stuttering behavior and hemispheric processing studies. Composites of left and right hemisphere processing resources over time may emerge in stutterers that differ from those of nonstutterers. Indeed, the composite configuration of left and right hemispheric resources for discrete segments of the processing epoch may provide us with critical information about stuttering.

EEG topography is a recently developed technique which provides a two-dimensional display of spontaneous and evoked potential activity of the brain recorded from multiple electrodes. Brain electrical activity mapping (Duffy, Burchfiel, & Lombroso, 1979) summarizes EEG and evoked potential data as color maps which can be cartooned into discrete segments across the recording epoch. Consequently, the investigator is able to study spatial and temporal patterns of ongoing brain electrical activity. This procedure has been useful in the delineation of regionally specific electrophysiological differences in patients with brain tumors (Duffy, Burchfiel, & Lombroso, 1979; Lombroso & Duffy, 1980; Duffy, 1982), in boys with dyslexia (Duffy, 1981; Duffy, Denckla, Bartels, & Sandini, 1980), in the identification of covert epilepsy (Lombroso & Duffy, 1980; Lombroso, & Duffy, 1982), and in the discrimination of schizophrenic patients from normal controls (Morihissa, Duffy, & Wyatt, 1983). These studies have reported hemispheric laterality differences for specific brain areas in dyslexic and schizophrenic subjects compared to normal controls, indeed, differences between normals and dyslexics were reported for the supplementary motor area (Duffy, 1981).

In addition to the visual display of spatial and temporal patterns, significance probability maps (Duffy, Bartels, & Burchfiel, 1981) can be constructed from EEG and evoked potential data. This procedure involves the calculation statistics to determine differences between homologous points for two groups, based on their mean and variance matrices. The comparisons can be calculated for any specific epoch length and used to delineate regions of maximal group separation as a preliminary statistical procedure. Fast Fourier Transform data from electrodes associated with epochs and regions of maximal separation can be submitted to other univariate and multivariate statistical procedures across groups, tasks, and conditions. The major advantage of the EEG topographic procedure resides in its ability to increase our knowledge of spatial and temporal pattern differences in brain electrical activity of various subject groups for a variety of stimulus and task conditions.

Although there is evidence of different cortical processing strategies, it cannot be assumed that the difference originates solely at the cortical level. Asymmetries at the subcortical level might trigger or facilitate different hemispheric activation patterns at the cortical level. Yeudall (1985) has suggested that cortical asymmetries in stutterers may reflect thalamic mechanisms. Thalamic mechanisms associated with specific alerting responses may play a role in hemispheric asymmetries (q.v., Fedio &

Van Buren, 1975; Ojemann, 1975; Ojemann, Fedio, & Van Buren, 1968).

Averaged evoked potential procedures may provide some information that provides insight into the role of attentional mechanisms in stutterers. The P300 is a late component of the human average evoked potential (Morsty, Duffy, & McCarley, 1983) that is elicited by linguistic or nonlinguistic stimuli (Kutas & Donchin, 1976), that are both surprising and relevant to the subject. The P300 appears to be associated with the orienting response (Friedman, 1976). The waveform is positive in amplitude and has a latency within the average range of 250 to 450 milliseconds. The P300 provides some index of the brain's response to changes in attended stimuli and may be generated from subcortical sources (Morsty, Duffy, & McCarley, 1983; Wood, Allison, Goff, Williamson, & Spencer, 1980; Halgren, Squires, Wilson, Rohrbaugh, Babb, & Crandall, 1980). Recent reports have found asymmetry of the P300 with normal subjects showing a greater P300 over the left hemisphere than over the right (Morsty, Duffy, & McCarley, 1983; Megela & Teyler, 1976).

Exploration of the P300 evoked potential may provide information with regard to the influence of attention on hemispheric processing in both stutterers and nonstutterers. Asymmetries of the P300 between groups could provide preliminary empirical support for subcortical involvement in stuttering and a base for future studies.

It has not yet been determined if anomalous hemispheric processing in stutterers is associated with atypical morphology of the cerebral hemispheres. The findings of Strub et al. (1987) which revealed atypical occipital asymmetries in two left-handed stuttering siblings are provocative. Continued investigation into atypical morphology using CT scans and nuclear magnetic resonance/magnetic resonance imaging (NMR/MRI) seems warranted. Imaging by nuclear magnet resonance is primarily suited to the imaging of soft tissue and is capable of producing sagittal, coronal, and transverse scans (Allen, 1984; Radda & Allen, 1983). Due to the high visual resolution, the lack of radiation, and the noninvasive method of the MRI procedure, it seems particularly well suited to the investigation of brain morphology in healthy subjects.

Although it is tempting to infer psychophysiological functions from morphological data it can also be inaccurate. The results from imaging studies alone will shed little light on the relationship between hemispheric activation and morphological asymmetries. Results from imaging studies must be correlated with hemispheric activation data from a variety of linguistic tasks with stutterers and nonstutterers before acceptable conclusions can be reached about the relationship of cerebral morphology to hemispheric processing. Yet, such investigation would add much to the development of a CNS model of stuttering.

There are several other areas that we must address including: (a) Language variables (q.v., Moore, 1984; Moore & Boberg, 1987) associated with increases in stuttering frequency relative to hemispheric activation; (b) the relationship of stuttering severity and hemispheric activation; (c) the relationship between motor speech deficits in stutterers and hemispheric activation; (d) hemispheric processing over the lifespan of stutterers (very limited data are available which addresses hemispheric activation in children who stutter) and (e) hemispheric processing of male and female stutterers; and the relationship of fluency management to hemispheric activation.

These are by no means easy areas of research inquiry. We need to consider carefully our conceptualization of brain-language relationships and the research methodology needed to answer the questions we propose. Nevertheless, with new techniques and knowledge available for the study of spatial and temporal aspects of brain electrical activity and brain imaging we stand on the threshold of a new appreciation of the neurophysiological mechanisms responsible for stuttered verbal behavior.

REFERENCES

ALLEN, P. (1984). In vivo applications of NMR to medicine. *Radiation, Physics, and Chemistry, 24,* 419–435.

BLOOD, G. W. (1985). Laterality differences in child stutterers: Heterogeneity, severity levels, and statistical treatments. *Journal of Speech and Hearing Disorders, 50,* 66–72.

BLOODSTEIN, O. (1987). *A handbook on stuttering.* Chicago: The National Easter Seal Society.

BOBERG, E., YEUDALL, L., SCHOPFLOCHER, D., & BO-LASSEN, P. (1983). The effects of an intensive behavioral program on the distribution of EEG alpha power in stutterers during the processing of verbal and visuospatial information. *Journal of Fluency Disorders, 8,* 245–263.

BRADY, J. P., & BERSON, J. (1975). Stuttering, dichotic listening and cerebral dominance. *Archives of General Psychiatry, 32,* 1449–1459.

CARUSO, A., ABBS, J., & GRACCO, V. (1988). Kinematic analysis of multiple movement coordination during speech in stutterers. *Brain, 111,* 439–455.

CERF, A., & PRINS, D. (1974). *Stutterers' ear preference for dichotic syllables.* Paper presented to the Annual Convention of the American Speech and Hearing Association, Las Vegas.

CIMORELL-STRONG, J. M., GILBERT, H. R., & FRICK, J. V. (1983). Dichotic speech perception: A comparison between stuttering and nonstuttering children. *Journal of Fluency Disorders, 8,* 77–91.

CURRY, F., & GREGORY, H. (1969). The performance of stutterers on dichotic listening tasks thought to reflect cerebral dominance. *Journal of Speech and Hearing Research, 12,* 73–82.

DAVENPORT, R. W. (1979). *Dichotic listening in four severity levels of stuttering.* Paper presented at the Annual Convention of the American Speech and Hearing Association, Atlanta.

DONCHIN, E., & MCCARTHY, M. (1979). Event-related brain potentials in the study of cognitive processes. In Ludlow, C. & Doran-Quine, M. (Eds.), *The neurological bases of language disorders in children: Methods and directions for research.* Bethesda: NIH Publication No. 79–440.

DORMAN, M. F., & PORTER, R. J., JR. (1975). Hemispheric lateralization for speech perception in stutterers. *Cortex, 11,* 181–185.

DUFFY, F. H. (1982). Topographic display of evoked potentials: Clinical applications of brain electrical activity mapping (BEAM). *Annals of the New York Academy of Science, 388,* 183–196.

DUFFY, F. H. (1981). Brain electrical activity mapping (BEAM): Computerized access to complex brain function. *International Journal of Neuroscience, 13,* 55–65.

DUFFY, F. H., BURCHFIEL, J. L., & LOMBROSO, C. T. (1979).

Brain electrical activity mapping (BEAM): A method for extending the clinical utility of EEG and evoked potential data. *Annals of Neurology, 5,* 309–321.

DUFFY, F. H., BARTELS, P. H., & BURCHFIEL, J. L. (1981). Significance probability mapping: An aid in the topographic analysis of brain electrical activity. *Electroencephalography and Clinical Neurophysiology, 51,* 455–462.

DUFFY, F. H., DENCKLA, M. B., BARTELS, P. H., & SANDINI, G. (1980). Dyslexia: Regional differences in brain electrical activity by topographic mapping. *Annals of Neurology, 7,* 412–420.

FEDIO, P., & VAN BUREN, J. (1975). Memory and perceptual deficits during electrical stimulation in the left and right thalamus and parietal subcortex. *Brain and Language, 2,* 78–100.

FITZGERALD, H., COOKE, P., & GREINER, J. (1984). Speech and bimanual hand organization in adult stutterers and nonstutterers. *Journal of Fluency Disorders, 9,* 51–65.

FRIEDMAN, D. (1976). The late positive component and orienting behavior. In D. Otto (Ed.), *Multidisciplinary perspectives in event-related brain potential research.* Washington, DC: U.S. Environmental Protection Agency.

GATES, A., & BRADSHAW, J. (1977). The role of the cerebral hemispheres in music. *Brain and Language, 9,* 403–431.

GOLDBERG, G. (1985). Supplementary motor area structure and function: Review and hypotheses. *Behavioral and Brain Sciences, 8,* 567–615.

GORDON, W. H. (1979). Left hemisphere dominance of rhythmic elements in dichotically-presented melodies. *Cortex, 15,* 58–70.

HALGREN, E., SQUIRES, N., WILSON, C., ROHRBAUGH, J., BABB, T., & CRANDALL, P. (1980). Endogenous potentials generated in the human hippocampal formation and amygdala by infrequent events. *Science, 14,* 803–805.

HAND, C. R., & HAYNES, W. O. (1983). Linguistic processing and reaction time differences in stutterers and nonstutterers. *Journal of Speech and Hearing Research, 26,* 181–185.

JENKINS, P., & MOORE, W. H., JR. (1985). The effects of visual feedback on hemispheric alpha asymmetries and reported processing strategies: A single-subject experimental design. *Brain and Cognition, 4,* 1985.

KENT, R. D. (1984). Stuttering as a temporal programming disorder. In R. F. Curlee & W. H. Perkins (Eds.), *Nature and treatment of stuttering: New directions.* San Diego: College-Hill Press.

KUTAS, M., & DONCHIN, E. (1976). Variations in the latency of P300 as a function of variations in semantic categorizations. In D. Otto (Ed.), *Multidisciplinary perspectives in event-related brain potential research.* Washington, DC: U.S. Environmental Protection Agency.

LIEBETRAU, R. M., & DALY, D. A. (1981). Auditory processing and perceptual abilities of "organic" and "functional" stutterers. *Journal of Fluency Disorders, 6,* 219–231.

LOMBROSO, C. T., & DUFFY, F. H. (1982). Brain electrical activity mapping in the epilepsies. In H. Akimoto, H. Kajamatsuri, & M. Seino (Eds.), *Advances in Epileptology: The 13th Epilepsy International Symposium* (pp. 173–179). New York: Raven Press.

LOMBROSO, C. T., & DUFFY, F. H. (1980). Brain electrical activity mapping as an adjunct to CT scanning. In R. Canger, F. Angeleri, & J. Penry (Eds.), *Advances in Epileptology: 11th Epilepsy International Symposium* (pp. 83–88). New York: Raven Press.

MEGELA, A. L., & TEYLER, T. J. (1976). Event-related potentials associated with linguistic stimuli: Semantic vs low-order effects. In Otto, D. *(Eds.), Proceedings of the Fourth International Congress on Event-Related Slow Potentials of the Brain.* Hendersonville, North Carolina.

MOORE, W. H., JR. (1986b). Hemispheric alpha asymmetries of stutterers and non-stutterers for the recall and recognition of words and connected reading passages: Some relationships to severity of stuttering. *Journal of Fluency Disorders, 11,* 71–89.

MOORE, W. H., JR. (1986b). Stuttered verbal behavior: A neurolinguistic perspective (Invited article). *Hearsay, A Journal of the Ohio Speech-Language-Hearing Association, Fall,* 74–79.

MOORE, W. H., JR. (1984a). Hemispheric alpha asymmetries during an electromyographic biofeedback procedure for stuttering: A single-subject experimental design. *Journal of Fluency Disorders, 17,* 143–162.

MOORE, W. H., JR. (1984b). Central nervous system characteristics of stutterers. In R. Curlee & W. Perkins (Eds.), *Nature and treatment of stuttering: New directions.* San Diego: College-Hill Press.

MOORE, W. H., JR. (1976). Bilateral tachistoscopic word perception of stutterers and normal subjects. *Brain and Language, 3,* 434–442.

MOORE, W. H., JR. (1979). Alpha hemispheric asymmetry of males and females on verbal and on-verbal tasks: Some preliminary results. *Cortex, 15,* 321–327.

MOORE, W. H., JR., & BOBERG, E. (1987). Hemispheric processing and stuttering. In L. Rustin, H. Purser, & D. Rowley *(Eds.), Progress in the treatment of fluency disorders.* London: Taylor & Francis.

MOORE, W. H., JR. CRAVEN, D. C., & FABER, M. (1982). Hemispheric alpha asymmetries of words with positive, negative, and neutral arousal values preceding tasks of recall and recognition: Electrophysiological and behavioral results from stuttering males and nonstuttering males and females. *Brain and Language, 17,* 211–224.

MOORE, W. H., JR., & HAYNES, W. O. (1980). Alpha hemispheric asymmetry and stuttering: Some support for a segmentation dysfunction hypothesis. *Journal of Speech and Hearing Research, 23,* 229–247.

MOORE, W. H., JR., & LANG, M. K. (1977). Alpha asymmetry over the right and left hemispheres of stutterers and control subjects preceding massed oral readings: A preliminary investigation. *Perceptual and Motor Skills, 44,* 223–230.

MOORE, W. H., JR., & LORENDO, L. (1980). Alpha hemispheric asymmetries of stuttering and nonstuttering subjects for words of high and low imagery. *Journal of Fluency Disorders, 5,* 11–26.

MORIHISA, J., DUFFY, F. H., & WYATT, R. (1983). Brain electrical activity mapping (BEAM) in schizophrenic patients. *Archives of General Psychiatry, 40,* 719–728.

MORSTYN, R., DUFFY, F. H., & MCCARLEY, R. (1983). Altered P300 topography in schizophrenia. *Archives of General Psychiatry, 40,* 729–734.

MOSCOVITCH, M. (1977). The development of lateralization of language functions and its relation to cognitive and linguistic development: A review and some theoretical speculations. In S. J. Segalowitz & F. Gruber (Eds.), *Language development and neurological theory.* New York: Academic Press.

OJEMANN, G. A. (1975). Language and the thalamus: Object naming and recall during and after thalamic stimulation. *Brain and Language, 2,* 101–120.

OJEMANN, G. A., FEDIO, P., & VAN BUREN, J. M. (1968). Anomia from pulvinar and subcortical parietal stimulation. *Brain, 91,* 99–116.

ORTON, S. T. (1927). Studies in stuttering. *Archives of Neurology and Psychiatry, 18,* 671–672.

PERRIN, K. L., & EISENSON, J. (1970). *An examination of ear preference for speech and nonspeech stimuli in a stuttering population.* Paper presented at the Annual Convention of the American Speech and Language Association, New York.

PETERS, R. W., LOVE, L., OTTO, D., WOOD, T., & BENINGUS, V. (1976). Cerebral processing of speech and non-speech signals by stutterers. *Proceedings of the International XVI Congress of Logopedics and Phoniatrics.* Basel: Karger.

PIENIADZ, J., & NAESER, M. (1984). Computed tomographic scan asymmetries and morphologic brain asymmetries-Correlations in some cases post mortem. *Archives of Neurology, 41,* 403–409.

PINSKY, S. D., & MCADAM, D. W. (1980). Electroencephalographic and dichotic indices of cerebral laterality in stutterers. *Brain and Language, 11,* 374–397.

PLAKOSH, P. (1978). *The functional asymmetry of the brain: Hemispheric specialization in stutterers for processing of*

visually presented linguistic and spatial stimuli. Unpublished doctoral dissertation, the Palo Alto School of Professional Psychology.

PONFORD, R., BROWN, W., MARSH, J., & TRAVIS, L. (1975). Evoked potential correlates of cerebral dominance for speech perception in stutterers and nonstutters. *Electroencephalography and Clinical Neurophysiology, 39,* 434.

PRESCOTT, J., & ANDREWS, G. (1984). Early and late components of the contingent negative variation prior to manual and speech responses in stutterers and non-stutterers. *International Journal of Psychophysiology, 2,* 121–130.

QUINN, P. T. (1972). Stuttering, cerebral dominance and the dichotic word test. *Medical Journal of Australia, 2,* 639–643.

RADDA, G., & ALLEN, P. (1983). Nuclear magnetic resonance techniques in medicine. *Annals of Internal Medicine, 98,* 514–529.

RAY, D., & MOORE, W. H., JR. (1988). *Hemispheric alpha asymmetries of stutterers during a quasi conversational task.* Paper presented at the Annual Convention of the American Speech-language-hearing Association, Boston.

ROLAND, P., LARSEN, B., LASSEN, N., & SKINHOJ, E. (1980). Supplementary motor area and other cortical areas in organization of voluntary movements in man. *Journal of Neurophysiology, 43,* 118–136.

ROSENFIELD, D. B., & GOODGLASS, H. (1980). Dichotic testing of cerebral dominance in stutterers. *Brain and Language, 11,* 170–180.

SHENKER, R., SEITZ, M., & DOEHRING, D. (1980). Stuttering behavior and cerebral asymmetry. In M. Seitz, B. Weber, J. Jocobson, & R. Morehouse, (1980). The use of averaged electroencephalic response techniques in the study of auditory processing related to speech and language. *Brain and Language, 11,* 261–284.

SOMMERS, R. K., BRADY, W., & MOORE, W. H., JR. (1975). *Perceptual and Motor Skills, 41,* 931–938.

STRONG, J. C. (1978). Dichotic speech perception: A comparison between stutterers and nonstutterers. *Asha, 20,* 728 (abstract).

STUSS, D. T., & BENSON, D. F. (1984). Neuropsychological studies of the frontal lobes. *Psychological Bulletin, 95,* 3–28.

STRUB, R., BLACK, W., & NAESER, M. (1987). Anomalous dominance in sibling stutterers: Evidence from CT scan asymmetries, dichotic listening, neuropsychological testing and handedness. *Brain and Language, 30,* 338–350.

THATCHER, R. W. (1980). Neurolinguistics: Theoretical and evolutionary perspectives. *Brain and Language, 11,* 235–260.

TRAVIS, L. E. (1931). *Speech pathology.* New York: Appleton-Century-Crofts.

VALSINER, J. (1983). Hemispheric specialization and integration in child development. In S. Segalowitz (Ed.), *Language functions and brain organization.* New York: Academic Press.

WEBSTER, W. G. (1988). Neural mechanisms underlying stuttering: Evidence from bimanual handwriting performance. *Brain and Language, 33,* 226–244.

WEBSTER, W. G. (1985). Neuropsychological models of stuttering-I. Representation of sequential response mechanisms. *Neuropsychologia, 23,* 263–267.

WEBSTER, W. G. (1986). Neuropsychological models of stuttering-II. Interhemispheric interference. *Neuropsychologia, 24,* 737–741.

WEBSTER, W. G., & THURBER, A. D. (1978). Problem-solving strategies and manifest brain asymmetry. *Cortex, 14,* 474–484.

WELLS, B. G., & MOORE, W. H., JR. (1988). *Linguistic variables and EEG alpha asymmetries in stutterers and nonstutterers.* Paper presented to the 1988 Annual Convention of the American Speech-Language-Hearing Association, Boston.

WILLIS, S., WHEATLEY, G., & MITCHELL, O. (1979). Cerebral processing of spatial and verbal-analytic tasks: An EEG study. *Neuropsychologia, 17,* 473–484.

WILKINS, C., WEBSTER, R. L., & MORGAN, B. T. (1984). Cerebral lateralization of visual stimulus recognition in stutterers and fluent speakers. *Journal of Fluency Disorders, 17,* 131–141.

WOOD, C., TRUETT, A., GOFF, W., WILLIAMSON, P., & SPENCES, D. (1980). On the neural origin of P300 in man. *Progress in Brain Research, 54,* 51–56.

WOOD, F., STUMP, D., MCKEEHAN, A., SHELDON, S., & PROCTOR, J. (1980). Patterns of regional cerebral blood flow during attempted reading aloud by stutterers both on and off haloperidol medication: Evidence for inadequate left frontal activation during stuttering. *Brain and Language, 9,* 141–144.

YEUDALL, L. T. (1985). A neuropsychological theory of stuttering. In E. Boberg (Ed.), *Seminars in speech and language: Stuttering, part two.* New York: Thieme-Stratton.

ZIMMERMAN, G. N., & KNOTT, J. R. (1974). Slow Potentials of the brain relation to speech processing in normal speakers and stutterers. *Electroencephalography and Clinical Neurophysiology, 37,* 599–607.

F. TREATMENT

Chapter 10

CURRENT TRENDS IN THERAPY FOR STUTTERING CHILDREN AND SUGGESTIONS FOR FUTURE RESEARCH*

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St. Louis and Westbrook (1987) have thoroughly and carefully reviewed current treatment options for stutterers. There is little to be done to elaborate or supplement this excellent work here. There have, however, been several recent trends in the treatment of children who stutter. The rest of this chapter will be devoted to the identification of those trends and to recommendations for research that will lead to better treatment for this population.

RECENT TRENDS IN THE TREATMENT OF CHILDREN WHO STUTTER

Early Intervention and Prevention

Certainly the most noteworthy trend in recent years has been the proliferation of early intervention programs. From the earliest days of our field until the sixties, it was considered potentially harmful to treat young stuttering children directly, and only parent counseling was advocated (Johnson, 1955). In the early 70s, some movement in the direction of including the child in the intervention process could be found. Egolf, Shames, Johnson, and Kasprisin-Burelli, (1972) described methods for modifying the interactions between parents and children, although the emphasis was still strongly on the parents. By the early 80s, three programs advocating therapy for preschool children were discernible from published accounts (Gregory & Hill, 1980; Ryan, 1971; Shine, 1980). By 1989, there were at least five more programs (Conture & Kelly, 1988; Culp, 1984; Mallard, 1988; Meyers, 1988; Pindzola, undated; Riley & Riley, 1979; Rustin & Purser, 1988; Starkweather & Gottwald, 1984; 1986a) designed to treat very young stuttering children or children believed to be at risk for stuttering. These more recent programs vary in the theoretical beliefs about stuttering on which they are based, and they naturally vary also in the nature of the treatment they prescribe. They share only two traits—(a) a belief that waiting to see if stuttering will disappear of its own accord runs greater risks than treating the child whose speech shows evidence of abnormal disfluency, and (b) reported rates of success that are higher than those reported for adult stuttering treatment programs. Assuming that these rates of success are no more inflated than those reported for adult treatment programs, the differences are impressive. Adult programs report a range of success from 50%–75% (Franken, 1988b; Webster, 1974), but few treatment programs for young children report anything less than 90%, and several report nearly perfect results—"98 per cent" (Gregory, 1986), "close to 100 per cent"[1] (Starkweather, Gottwald, & Halfond, 1990). There is also a sharp difference in two other significant factors—the quality of speech in the treated person and the relapse rate. Relapse rates for treated adults are reported around 50% (Franken, 1988b; Webster, 1974), but those for children are reported to be close to zero (Gregory, 1986; Starkweather, Gottwald, & Halfond, 1990). Perhaps more importantly, adults who have successfully completed treatment have either carefully monitored speech (Boberg & Kully, 1985, pp. 5–7) with attendant diminished quality of speech (Franken, 1988b) or they have residual stuttering behaviors, which, however mild and acceptable to the individual, are nonetheless noticeably different from the disfluencies of nonstutterers (Prins, 1984, pp. 411–17). Although the earliest reports of the effects of treatment on very young children were similar to those for adults (Gregory & Hill, 1984; Shine, 1977), contemporary reports are strikingly different. Starkweather, Gottwald, and Halfond (1990), Got-

*This paper was prepared in part while the author was on a Fulbright Fellowship attached to the University of Nijmegen, The Netherlands.

[1]It should be noted that since this publication, one child has been encountered who failed to achieve normal speech in the program.

twald and Starkweather (1984), Starkweather and Gottwald (1986a), Gregory (1986) and Pindzola (undated) report essentially no difference between the speech of the preschool stutterers they have treated successfully and that of nonstuttering children.

Another feature of these contemporary early intervention programs is the relatively brief duration of treatment. Treatment duration is not always reported, but when it is, the calendar duration of treatment is likely to be 1–3 months, and the clock duration roughly 20 hours (Starkweather & Gottwald, 1986b). Although these figures are much shorter than treatment duration for adults, which range from 4 weeks (~140 hours) (Van Riper, 1973; Webster, 1974) under the most intense circumstances to many years. Treatment duration with preschool children is not, however, directly comparable with treatment duration of adult stutterers, because it is common for the parents of preschool children to be intensely involved in their children's treatment, both in the clinic and at home. This extends the intensity of treatment to almost 100% of the child's time, a level of intensity that is not usually possible with school-aged children and adults. One interesting aspect of treatment duration is its relation to age at onset. Starkweather and Gottwald (1986b) assessed the extent to which treatment duration could be predicted from information obtained at intake. They found that the amount of time that elapsed between the onset of stuttering as reported by the parents and the beginning of treatment was the most potent predictor of treatment duration ($r = .72$), indicating that the longer the "waiting time" between onset and treatment, the longer the treatment will take before the child is able to speak normally.

The Assessment of the Child's Communicative Environment

The theoretical view that stuttering in children changes in form and severity as a consequence of interactive processes between the stutterer and the communicative environment (Meyers & Freeman, 1985a; 1985b; Starkweather, Gottwald, & Halfond, 1990) has led to a number of trends in clinical practice. One such trend is the assessment at intake of a child's communicative environment, specifically the measurement of environment variables that are known or presumed to be related to the fluency of children. Starkweather, Gottwald, and Halfond (1990) describe assessment techniques that included measures of parents' speech rate, number of questions, number of interruptions, nonverbal reactions, and level of semantic and syntactic language use. Conture and Caruso (1987) and the Rileys (1979) described similar measures.

Intensive Treatment via Parental Involvement

A second trend in clinical practice is the increased involvement of parents in the therapy process. The idea of involving parents in the treatment of stuttering is hardly new, but recent years have been a sharp upturn in the extent of parental involvement and in the degree to which success in the program is felt to depend on the amount and quality of parental participation. Rustin and Purser (1988) attribute the success of their program in Great Britain to the inclusion of parents in the program, and Mallard (1988) echoes this opinion in his exposition of the American version. Starkweather, Gottwald, and Halford (1990) stress the importance of the parents' group in addition to individual parent counseling in the success of their early intervention program. The inclusion of parents as adjunct therapists in the home makes it possible to extend treatment to virtually every moment of a preschool child's life.

The trend to involve parents more than before is very clear in early intervention programs. The trend is less widespread in the school-aged population, despite arguments of Mallard (1988) and Rustin and Purser (1988). In time, it is hoped that this trend will become stronger.

Combinations of Environmental and Behavioral Management

Most of the new programs of early intervention combine direct management of the child's fluency behavior with attempts to alter the child's environment in ways that will facilitate the development of fluency. Although both behavioral and environmental management have been described from the earliest treatment programs, more and more of the current programs stress the value of using both approaches simultaneously (Conture & Kelly, 1988; Mallard, 1988; Starkweather, Gottwald, & Halfond, 1990).

Increased Awareness of the Role of Language

A current trend that is quite separate from the proliferation of early intervention programs is the increased awareness of language development and its possible relation to stuttering. Several relations can be identified. Certainly, there is ample clinical evidence that a common pattern of stuttering onset/development involves delayed language development. Specifically, many children begin to stutter during therapy for language delay. Several other findings seem to be related to this: Stuttering is more common in children with delayed language (including phonology) than in children without these disorders (Hall, 1977), and similarly, stuttering children are inclined, on the average, to be delayed slightly in language development (Andrews et al., 1983). Merits-Patterson and Reed (1981) showed quite clearly that it was children in therapy for delayed language development, not children with delayed language development waiting for treatment, who were more likely to develop stuttering. So it seems clear that it is the therapy more than the language delay itself that is the problem. Interestingly, when therapy for language delay is based on teaching parents to

talk more slowly and use simpler language forms (Goorhuis-Brouwer, 1987), the tendency for stuttering to begin during language therapy was absent in over 60 cases (Goorhuis-Brouwer, 1989).

SUGGESTIONS FOR FUTURE RESEARCH AND ANTICIPATED OBSTACLES TO PROGRESS

Assessment

There is a strong need in clinical assessment to rely less on the simple frequency count of stuttering behaviors and to include also some measure of the duration of stuttering behaviors. The suggestion to go beyond frequency counts is not new (Cooper, 1986). Although reliable, simple frequency counts of stuttering behavior may omit information that is of value in understanding the nature and severity of a child's problem. It seems obvious that the stutterer whose behaviors take up more time is more severely impaired than one whose behaviors are brief in duration. Yet duration is rarely measured in the clinic or laboratory. There appear to be two reasons for this, one historical and one practical.

The historical reason is, apparently, the discussion of duration in Bloodstein (1987). At least this discussion is typically cited in arguments against the measurement of stuttering duration. Bloodstein noted, citing several experimental investigations, that the duration of stuttering was not correlated with the frequency of stuttering. He noted also that the vulnerability of duration was very small, ranging from less than half a second to more than four seconds. It seems evident on the face of it, that variability in the duration of speech that ranges from 500 to 5000 msec is anything but small. 500 msec is the equivalent of 2.5–3 syllables of speech, while 5000 msec is equal to a sentence that is 25–30 syllables in length. The standard deviation of stuttering duration, reported as .75 sec (Sheehan, 1974) suggests a variability in speech time of about 3 syllables, which cannot easily be considered a "small" amount of variability. If it is given that the variability of stuttering duration is not small, then the low correlations with stuttering frequency that have been reported can be attributed to the independence of the two measures. If the measures are independent, then they should both be included in assessments of stuttering severity.

One way to incorporate duration with frequency is by measuring the proportion of a child's speech time that is taken up by stuttering behavior (Starkweather, Gottwald, & Halfond, 1990). This has several advantages. First, it is intuitively more closely related to the severity of the disorder than frequency counts. This becomes apparent in reports to laymen. When a clinician reports to a physician or teacher or parent that 40% of a child's speech time is spent in stuttering behavior, it is very clear how severe the problem is—much more so than a report that the child stutters on 5% of his words. It is not just that the numbers are bigger, but rather that they express more clearly the nature and severity of the problem. Second, when the proportion of speech time spent on stuttering is assessed individually for each type of speech behavior, it is possible to assess accurately the relative contribution of different behaviors to the problem. A child who spends 5% of his speech time on repetitions and 40% of his speech time on revision should probably tackle the revision first. It will have a bigger impact on his ability to communicate effectively.

The duration of stuttering behavior is, without question, a more valid measure of the severity with which the disorder impairs communication than the frequency of behavior. Stuttering impairs communication by occupying time that in normal speakers contains meaningful utterance. The difficulty with the measurement of duration lies in the method of measurement. Time is fleeting and capturing it difficult. Conture and Caruso (1986) suggest the use of a stopwatch for this purpose. Although the stopwatch has the important characteristics of convenience and inexpensiveness, it unfortunately lacks precision. There is a delay between the onset of the stuttering behavior and the clinician's response, attributable to the clinician's reaction time. One might expect this delay to be cancelled by an identical reaction time before stopping the watch in response to the offset of the behavior, but unfortunately the two are not equal, the reaction time to offset being longer, which tends to overestimate the duration of behaviors by an amount equivalent to the difference between the onset and the offset reaction times. This varies somewhat from clinician to clinician, and from sample to sample, so that an accurate mathematical correction is not totally possible. And of course, the error factor has a much bigger effect on the brief behaviors, which are by far the most frequently occurring. Probably a better measure is the use of a clock with a 10-second sweep hand. By looking at the clock and noting the point where the hand is on the dial at the times when the stuttering behavior begins and ends, it is possible to determine the duration of the behavior. This method seems a little better, and is also inexpensive, but it requires somewhat more training to achieve a reliable level of skill. In any event, no formal comparisons have been made.

The measurement of acoustic durations can be made with great precision and reliability by using a computer and an acoustic analysis program that permits simultaneous sound and visual inspection of the waveform (Crystal & House, 1988). These are available but neither cheap nor convenient. Consequently, a study comparing clinically feasible methods of duration measurement with highly accurate methods would be useful.

A useful way to approach this experiment would be to compare the different measures through three stages of stimulus complexity. In the first stage, a set of nonspeech stimuli, pure tones for example, would be used, including a wide variety of known durations. The clinically feasible methods under consideration could then be compared (e.g., stopwatch vs. sweep-hand vs. computerized graph-

ics, using a cadre of competent clinical speech pathologists as subjects). Next, a sampling of speech stimuli, utterances produced by normal children and adults, also of varying known durations, but free of disfluency, could be used as stimuli and the methods compared once again. Finally, a set of samples of speech utterances containing disfluencies, both normal and abnormal, could be used as stimuli. In this way the influence of stimulus-complexity and context on measurement will be understood.

There are other questions about the measurement of duration that also need to be addressed. Although stuttering duration seems clearly to be related to severity, we need to know how it relates to other measures of importance. Chief among these are the measures of speech motor control, speech rate, reaction time, measures of motor programming, and measures of motoric execution, which have been shown repeatedly (Borden, 1983; Peters, Hulstijn, & Starkweather, 1989; Watson & Alfonso, 1987) to distinguish sharply between stutterers and nonstutterers and between different levels of stuttering severity.

Consequently, a comprehensive study of stuttering durations would be of substantial value. First, we need to identify the best practical method for measuring it, then we need to establish standardized norms for the amount of time normal children of various ages spend on disfluent speech as a proportion of their speech time, and finally we need to determine the relations between the new measure and other important measures.

The Effectiveness of Early Intervention Programs

Outcome assessment. Various early intervention programs for stuttering have been developed and refined. It seems appropriate now to evaluate them. Several things need to be considered in undertaking the evaluation of early intervention programs. First, the therapies differ from one another in several dimensions yet share some common characteristics. It would perhaps be most useful to evaluate specific techniques as well as entire therapeutic packages. In this way the contribution of each technique and the unique combination of techniques that defines a specific program can both be evaluated. One technique, used by many of these early intervention programs—training parents to talk more slowly to their children—has already been evaluated. Stephenson-Opsal and Bernstein-Ratner (1988) have shown that the reduction of parental speech rate is effective in decreasing the frequency of disfluency in preschool children.

Other specific indirect techniques, such as parental language simplification, question reduction, interruption reduction, demand speech reduction, parental modeling of disfluencies, the "fluency-enhancing environment" remain to be tested. Some direct techniques are widely used in programs for adults (e.g., rate reduction, easy onset, blending, pull-out, sound prolongation, voluntary stuttering, and contingency management), and some of the programs have reported outcome data, but, with the exception of contingency management, there is little data on effectiveness for specific techniques, even with adults.

In light of the claims for success made by the proponents of early intervention programs, a high standard of outcome should be used. The therapies have in general claimed to result in normal, not controlled, speech, to have reached this level of speech performance in a relatively short period of time, and to have produced results that are more resistant to relapse than therapies for adults and older children. In addition, the tendency for children at this age to recover "spontaneously," however inflated it may have been by earlier reports (Ingham, 1984), seems nonetheless to be a real tendency. For this reason also, a high standard of success should be employed. Franken (1988a) has devised a set of scales with which to evaluate adult therapy outcome that are based on criteria for speech excellence in nonstutterers. The adaptation of these scales for use with children would be a useful research goal.

It would also be of great interest to assess the effectiveness of a given therapeutic approach on children who come into therapy at different ages post-onset, since other, less objective, data suggests that therapy will be more effective the sooner it begins (Starkweather & Gottwald, 1986b).

A major barrier to assessing the outcome of treatment programs for children is the fact that fluency changes over time. In most children, fluency improves, but in others it deteriorates into stuttering. As a result, it can be difficult to sort out developmental from therapeutic change. For this reason, it is desirable to gain a better understanding of the variables that influence normal fluency development. Considerable insight can be gained when these variables are considered within the broader context of general child and family development. Consequently, studies that seek information about the development of the elements of fluency—continuity, rate, coarticulation, rhythm, and effort—as they influence and are influenced by the changing speech behavior of other family members should be supported.

To the extent it is feasible, these different elements of fluency should be studied simultaneously, so that information about their interaction can be gained. Consequently, a multivariate study is suggested as a place to begin. Once some understanding of the relationships among fluency variables is attained, then more univariate studies can be undertaken with a better chance of being interpreted correctly. Both longitudinal and stratified experiments are useful. Longitudinal studies have the advantage of controlling for the important age × subject interactions, but stratified samples, in addition to producing results more quickly, also control for cultural factors, which can change over time and influence experimental variables in much the same way as development.

An important consideration in studies of normal fluency is the tendency for gene pools to be restricted to specific geographical areas, particularly where movement into and out of a community is restricted. Because we know that stuttering is influenced by genetic background (Kidd 1980, inter alia), it follows that normal fluency may be

also. This suggests that importance, in studies of normal fluency development, of selecting subjects from a wide geographical area.

Outcome Prediction (Prognosis)

Two methods for predicting the outcome of therapy seem likely to provide information that will prove useful in evaluating the effectiveness of early intervention programs. The first method is to assess the correlation of individual items of information taken at intake with a chosen criterion of therapeutic success. The criterion might be the extent of therapeutic change (pre-post differences in fluency level) or the duration of treatment. Gottwald and Starkweather (1986a) carried out a preliminary study of this sort in an effort to refine an existing program of early intervention. They performed at intake a series of measures which they suspected of having predictive value. Therapy was then carried out until the children were speaking normally. Finally, the correlations between treatment duration and the suspected predictive measures was calculated. Three suggestive results were obtained: (a) There was a strong relationship between the time from stuttering onset to therapy intake and the duration of therapy, which was mentioned earlier. (b) There was also a pattern of correlations between parental behavior and children's fluency that suggested the importance of parental reactions in both the development and the recovery of stuttering. (c) There was a very strong relationship between the amount of change in parents' speech rate and the amount of change in the child's fluency.

A more extensive study of this sort, or a variation of it, would be useful in making clear what aspects of therapy are the most important. A regression analysis, instead of a simple correlation matrix, would of course provide information about the relative contributions of the various intake measures to the prediction of therapy duration. Also, there is no reason why other measures of therapeutic success cannot be used as criterion measures. Of course, prediction is not the same as control, but control is not always an option. Withholding treatment raises ethical questions which can not always be resolved. In some cases, where a waiting list is unavoidable, the control group can be taken from the waiting list and then, when treated, can serve as the treatment group. This has considerable experimental value. But in some clinical settings, often encountered in early intervention programs, the waiting list itself is seen as highly undesirable because of the rapid deterioration in fluency level that can occur in this group. As a result, control is often simply not an option, and studies of prediction are the next best alternative.

Studies of prediction can also be seen from another perspective—they assess the value of information taken at intake, since items that do not predict outcome may not be useful. The eventual goal of such a study is to answer two questions: (a) what information to gather and (b) how to use it to prepare a therapy plan that will be maximally efficient. At the moment clinicians feel uncertain about how to make these decisions. They are afraid to omit a procedure or a technique. Old habit, and the precedent of earlier practices count more heavily in these decisions because of uncertainty over what can be predicted from diagnostic information. The result is defensive lesson plans—trying everything in the hope that something will be effective—and it is not good clinical practice. It would better for our clients, saving them time and money, if these decisions could be based on a clearer understanding of what each item of diagnostic information means with regard to therapy planning.

There are several aspects of outcome that would be useful to predict. The first, of course, is the fluency level of the child, and the fluency could be defined in a number of different ways, depending on the theoretical stance of the experimenter. It would also be useful to predict the extent of changes in parental speech. Many therapies seek reductions in parental speech rate, language level, talkativeness, demand speech, nonverbal reactions to stuttering, questioning, interruptions, etc., and clinicians would approach therapy with a better understanding of its difficulty if they could know, at the time of intake, the probability of making these changes successfully.

A particularly important prediction is the probability of relapse. Clinical accounts suggest that relapse is a minor, almost nonexistent problem for the school-age child. A study of relapse prediction for children of various ages would be able to document these clinical accounts more clearly and empirically. More importantly, it would lead to a better understanding of the process of relapse. Clearly it would be useful to be able to determine at the onset of therapy what the probability of relapse is, and a predictive study might even suggest what to do about it. Some of the British work (Rustin & Purser, 1988) indicates that parental involvement is important in preventing relapse in school-aged children, but a more complete understanding of the items that predict relapse will surely be even more useful. It looks as though one reason for relapse in school-aged children is that stuttering has already become chronic by the time a child reaches school age, but the means for dealing with chronic stuttering are more effective with older stutterers. School children seem able to learn motor control techniques quickly and easily, but they abandon them more quickly. We know many ways to minimize relapse (Boberg, 1981), but I don't think we really know what happens when a stutterer relapses. I think a study in which the process of relapse was closely monitored, preferably at the physiological level, would be useful. Such a study should probably be undertaken with adults first, but the implications for the child, at least the school-aged child, would be substantial.

As a suggestion, one way of monitoring relapse closely would be to study a similar phenomenon—the return of stuttering behaviors after fluency-enhancement procedures have been withdrawn. This is an unexplored area with possible implications for the process of relapse. When fluency is induced artificially by choral speech, for example, and then the support speaker's voice is gradu-

ally withdrawn, stutterers can typically continue speaking with perfectly normal uncontrolled speech for a brief period of time (10 seconds to a minute in my experience). Research which carefully monitors the physiology of this minirelapse might be instructive for the larger problem of post-therapy relapse. Such an investigation would surely tell us something about the physiological events that accompany the difficulty that stutterers have in maintaining fluency that has previously been established.

The Language Connection

It seems quite evident that there is a relationship, or relationships, between language skill or language use and the development of stuttering (Starkweather & Gordon, 1983). The nature of this relationship is not clear, however, and the implications for therapy are not known, even though many clinicians either incorporate parental language simplification as an aspect of therapy for preschoolers (Starkweather, Gottwald, & Halfond, 1990) or structure therapy for both younger and older children around language variables (Wall & Meyers, 1984).

The complexity of the language issue can perhaps be appreciated by noting a few facts. First, there is good evidence that, on the whole, children who stutter are slightly delayed in their language development (Andrews et al., 1983) and that there are more stuttering children among the language delayed (Hall, 1977). But stuttering also develops in children whose language is advanced (Bloodstein, 1987). Amster (1989), reviewing cases of stuttering development in these children, concludes that there is a pressure to perform linguistically that the superior child experiences in much the same way as the child in therapy for language delay. This suggests the possibility of studying the effect of pressure to produce more complex language, or even just longer sentences, on the fluency of normal children. Gordon, Luper, and Peterson (1986) showed that the tendency for increased disfluencies to occur on more complex sentences in normal children occurs only when the children have to formulate the sentences and not when they simply have to repeat them, but a study designed to test the effect of performance pressure on disfluency would be useful. It would be particularly useful if, in such a study, the pressure were induced by positive reinforcement for longer or more complex sentences, which appears to be the way the parents of linguistically superior children induce it (by showing delight in their children's performance). The effect of such a contingency on the frequency and topography of normal disfluencies would be valuable to know, but the value of such a study would be greatly enhanced if physiological measures of muscle activity levels and the timing of speech movements could also be obtained by noninvasive methods.

The Critical Period

Andrews and Harris (1964) describe 43 cases of stuttering. In 25 cases the disorder began before age 5, 15 of whom recovered within 6 months. Similarly, there is a tendency, less clearly seen in the Andrews and Harris data, for stuttering that begins in the later school years to end within 6 months. There were 7 cases that began after age 6, of which 5 recovered within 6 months. Although it seems clear that the percentage of those who recover spontaneously is much lower than previously thought (Ingham, 1976; 1983), there is still a tendency for spontaneous recovery in children. But this tendency is not distributed evenly throughout the age range, nor does it progressively diminish. Instead, it appears that stuttering lasting for less than one year in the Andrews and Harris data is found in children younger than 5 (most) or older than 7 (a few). All but one of the children whose stuttering persisted began to stutter and were still stuttering during the period between 4 and 6. The one exception was a child whose stuttering began at 6 and persisted to the end of the study.

These data suggest that there may be a kind of critical period. If stuttering is present during this period (either because it began during the period or began earlier and persisted into the period), it is likely to persist. It may or may not be coincidental that dialectal/phonological patterns are also flexible before the same age (roughly seven) and become less flexible with increasing age beyond seven (Krashen, 1973). Data from treatment programs (Starkweather, Gottwald, & Halfond, 1990) seem to confirm this idea in that children over 6 require more time-consuming treatment.

Although these observations are only suggestive, the possibility of a critical period during which stuttering becomes chronic and/or less treatable has profound implications for the treatment of stuttering in children. Studies designed to confirm or deconfirm the critical interval concept would be useful. The obvious design to answer such a question is a longitudinal study of stuttering children in which the time of onset and the duration of the disorder are very carefully documented. One roadblock to the success of such a study is that treatment effects may confound the results. A careful documentation of accompanying treatment procedures will alleviate, but not solve, this problem.

However, another useful approach is through studies of fluency development in normal children. A longitudinal study of normal fluency development, mentioned above, designed to address the critical period hypothesis, would be useful. For example, researchers could test the changeability of various aspects of speech production at different ages in the longitudinal sample. Variability in the duration of sounds in younger children has been demonstrated (Kent & Forner, 1980). But it is not clear if this variability represents a lack of speech motor control, or a flexibility in speech motor control systems which allows the system to be changed. A direct test of changeability at different ages, applied to a variety of parameters (e.g., speech and duration, rate, stress patterns, would provide some information relevant to the critical period hypothesis).

The Speech Motor Skills of Children

A substantial amount of recent work (Guitar et al., 1988; Peters et al., 1989; van Lieshout et al., 1988; Watson & Alfonso, 1987a) indicates abnormalities in the speech motor output of stutterers. Most of these data have been interpreted as suggesting that there are deficits in stutterers' speech motor control systems. However, it is also reasonable to interpret these results as indicating an acquired deficit, nature unspecified, that results in deviant output. In other words, the motor output deviations may result from the disorder rather than be a reflection of its etiology. Similar studies of children—based on reaction time or on other paradigms—will shed light on this problem, but not resolve it entirely because of the fact, now well established Schwartz and Conture (1988) that a healthy minority of stuttering children, even some very young children, show symptoms of struggle, tension, avoidance, and other typical signs of "advanced" stuttering. The most reasonable explanation of this pattern seems to be that at least some of the motor disturbances of stutterers, as reflected in higher EMG amplitudes, longer EMG durations, and mistimed EMG onsets, may be attributed to the development, rather than the etiology, of the disorder.

Thus, motoric disturbances may be both cause and effect, suggesting a circular pattern. Two recent results may be attributed to such a pattern. Hubbard and Yairi (1988) showed that stuttering children's disfluencies are significantly more clustered than those of nonstuttering children, and in stutterers the clusters tend to be of longer duration. This suggests compellingly that at least some aspects of abnormal disfluency are caused or precipitated by disfluency itself, normal or abnormal. Similarly, Schwartz and Conture (1988) developed subgroups of stuttering children based on behavioral characteristics. They demonstrated that associated behaviors are common in young stutterers and concluded that young stutterers may be reacting to their own stuttering.

Data showing motoric deficits in stutterers can thus be interpreted in several ways (Peters, 1987). The most theoretically vigorous interpretations suggest that stutterers may lack a fundamental ability to move the speech mechanism at velocities and with precision that is adequate for speech (Kent, 1983; Zimmermann, 1980). The most conservative interpretations suggests that stutterers may have learned to approach speech with effort and struggle of which unusual activity is a necessary adjunct (Bloodstein, 1987; Starkweather, 1987).

The lack of resolution of this profoundly fundamental issue has equally important implications for the treatment of children who stutter. If the motoric abnormalities of stuttering children are etiological, then we will need to develop new therapies that help even very young children learn to control and coordinate their speech mechanisms more effectively. If the motoric abnormalities are secondary, then we need to feel less timid about modifying the environments of very young stutterers and modifying the attitudes and emotional reactions of older children. If stuttering is a result of some combination of these two processes, then combinations of therapeutic approaches should be used to the exclusion of one-sided approaches of either kind.

Although a complete resolution of this controversy seems less than easy, data bearing on the issue could be obtained by an investigation of young stutterers designed to assess the relation between motor deficits, however defined (e.g., reaction time effects such as foreperiod effects, rhythm tracking, EMG timing and amplitude, EMG power spectra, variability of segment durations) and reaction to stuttering. The roadblock to such a study is the absence of any clear, operational definition of "reactive stuttering." This seems like a serious problem, in light of the fact that virtually every theorist acknowledges that reactions to stuttering play some role in the disorder, although some may see them as minimal.

It seems reasonable, however, that if some stuttering behaviors are attempts to minimize disfluency (e.g., behaviors such as word-changing, jaw-jerks that terminate tremor, backing up and restarting, timers, etc.) while others are not, a paradoxical intention test should sort them out. In other words, if stutterers are motivated, by instruction or an appropriate contingency, to stutter more, behaviors they have used to minimize stuttering should diminish, whereas when they are motivated, by instruction or an appropriate contingency, to stutter less, these same behaviors should increase. Specifically, *stuttering* as defined by the subject is first punished, then reinforced, and the frequency of specific behaviors is noted before, during, and after the contingency. Those behaviors that the person uses to minimize stuttering should decrease in frequency under conditions in which the person is attempting to maximize stuttering, or at least not trying so hard to minimize it. Those behaviors that the person defines as stuttering will change too, in the opposite direction, according to the extent to which the person's reactive behaviors are successful in minimizing stuttering. Such a double-barrelled paradoxical intention test should be developed for use with adults as well as with children. In addition to its value in helping to resolve the theoretical issue described above, it will have substantial clinical use in providing information that will help clinicians tailor therapy plans to fit the needs of individual stutterers.

Once an operational definition of reactive stuttering has been developed and verified, then it will be possible to gather data that will bear on the more fundamental issue described earlier—whether the motor deficits are cause or effect or both.

The Effects of Positive Emotion

There is virtually no empirical evidence that relates emotional states in children to stuttering, yet every clinician knows that most stuttering children will be less fluent under conditions of intense excitement, such as a birthday party or a visit from grandparents, as well as conditions of anxious uncertainty, such as the beginning

of school or when the family moves. Further, most stuttering children respond, often dramatically, to a "fluency enhancing environment" which is achieved by talking and interacting to the child in certain specific ways (Starkweather, Gottwald, & Halfond, 1990) that seem to reduce social anxiety and time pressure on the one hand, but also reduce positive excitement. Further, the observation that excitement disrupts fluency as much as anxiety if not more so (Adams, 1989), although not well established empirically, suggests rather strongly that the role of emotion in stuttering is not etiological but precipitatory. Because motor performance deteriorates under conditions of both anxiety and excitement and is improved under calmer emotional states, it seems likely that the effects of emotion on stuttering children may occur via an alleviation of motor disturbance.

Therefore, research designed to assess the effects of excitement on speech and non-speech motor behavior in normal children should be encouraged. An empirical verification that the reduction of positive excitement promotes fluency, will result in a sharp change in the therapies administered by a number of clinicians who currently see anxiety reduction as the major ingredient of therapy for children, accomplished through environmental manipulation in early intervention programs and through desensitization techniques in school-aged children. Investigations designed to verify these phenomena would be useful in understanding the development of stuttering in children and in providing direction in the building of therapy programs.

REFERENCES

ABBEDUTO, L. (1985a). *Syntactic, semantic, and rhythmic influences on children's and adults' motor programming of speech.* Unpublished manuscript, Vanderbilt University.

ABBEDUTO, L. (1985b). The effects of linguistic complexity on children's and adults' motor programming of speech. *Language and Speech, 28,* 361–75.

ALLEN, G. D., & HAWKINS, S. (1980). Phonological rhythm: Definition and development. In *Child phonology,* Vol. I: *Production.* New York: Academic Press.

AMSTER, B. (1984). The rate of speech in normal preschool children. Ph.D., Dissertation, Temple University.

ANDREWS, G., CRAIG, A., FEYER, A-M., HODDINOTT, S., HOWIE, P., & NEILSON, M. (1983). Stuttering: A review of research findings and theories circa 1982. *Journal of Speech and Hearing Disorders, 48,* 226–46.

ANDREWS, G., & HARRIS, M. (1964). The syndrome of stuttering. London: Heinemann.

BANDURA, A. (1969). Principles of behavior modification. New York: Holt, 1969.

BERLIN, C. I. (1960). Parents' diagnoses of stuttering. *Journal of Speech and Hearing Research, 3,* 372–79.

BLOODSTEIN, O. (1987). *The handbook of stuttering,* (4th edition). Chicago: National Easter Seal Society.

BLOODSTEIN, O., & GANTWERK, B. F. (1967). Grammatical function in relation to stuttering in young children. *Journal of Speech and Hearing Research, 10,* 786–789.

BOBERG, E. (1981). *Maintenance of fluency.* New York: Elsevier.

BOBERG, E., & KULLY, D. (1985). Comprehensive stuttering program. San Diego: College-Hill Press.

BORDEN, G. (1983). Initiation versus execution time during manual and oral counting by stutterers. *Journal of Speech and Hearing Research, 26,*

BRUTTEN, G. J., & TROTTER, A. (1986). A dual-task investigation of young stutterers and nonstutterers. *Journal of Fluency Disorders, 11,* 275–284.

BRUTTEN, G. J., & TROTTER, A. (1985). Hemispheric interference; a dual task investigation of youngsters who stutter. *Journal of Fluency Disorders, 10,* 77–85.

CONTURE, E., & CARUSO, A. (1987). Assessment and Diagnosis of Childhood Dysfluency. In L. Rustin, H. Purser, & D. Rowley (Eds.), *Progress in the treatment of fluency disorders.* London: Taylor & Francis.

CONTURE, E., & KELLY, E. (1988). *Remediation of stuttering in young children.* The Oxford Dysfluency Conference, Oxford, England.

COOPER, E. (1986). Treatment of disfluency; future trends. *Journal of Fluency Disorders, 11,* 317–27.

COOPER, E., & COOPER, C. (1985). Cooper personalized fluency control therapy revised. Allen, TX: DLM Teaching Resources.

CULP, D. (1984). The preschool fluency development program: Assessment and treatment. In M. Peins, (Ed.), *Contemporary approaches in stuttering therapy.* Boston: Little Brown.

DALY, D. (1984). Treatment of the young chronic stutterer: Managing stuttering. In R. Curlee, & W. Perkins (Eds.), *Nature and treatment of stuttering: New directions.* San Diego, CA: College-Hill Press.

DELL, C. (1980). *Treating the school-aged stutterer.* Memphis, Speech Foundation of America.

DISIMONI, F. G. (1974). Influence of utterance length upon bilabial closure duration of /p/ in three-, six-, and nine-year-old children. *Journal of the Acoustical Society of America, 55,* 1919–21.

EGOLF, D., SHAMES, G., JOHNSON, P., & KASPRISIN-BURELLI, A. (1972). The use of parent-child interaction patterns in therapy for young stutterers. *Journal of Speech and Hearing Disorders, 37,* 222–32.

FRANKEN, M. C. (1988a). *An instrument for the perceptual and acoustical evaluation of stuttering therapy.* Convention address, ASHA.

FRANKEN, M. C. (1988b). *Long-term follow-up of Webster's Precision Fluency Shaping Program in Stuttering.* Convention address, ASHA.

GOORHUIS-BROUWER, S. (1987). *Leren praten gaat niet helemaal vanzelf.* Groningen: Akademisch Ziekenhuis.

GOORHUIS-BROUWER, S. (1988). *Personal communication.*

GORDON, P., LUPER, H., & PETERSON, H. (1986). The effects of syntactic complexity on the occurrence of disfluencies in 5 year old children. *Journal of Fluency Disorders, 11,* 151–64.

GOTTWALD, S., & STARKWEATHER, C. W. (1984). *Stuttering prevention: Rationale and method.* Short course, ASHA.

GREGORY, H., & HILL, D. (1984). Stuttering therapy for children. In W. Perkins (Ed.), *Stuttering disorders.* New York: Thieme-Stratton.

GREINER, J. R., FITZGERALD, H. E., & COOKE, P. (1986). Speech fluency and hand performance on a sequential tapping task in left- and right-handed stutterers and nonstutterers. *Journal of Fluency Disorders, 11,* 55–69.

GUITAR, B. (1982). *Parent-child interactions: A case study in stuttering.* Unpublished manuscript, University of Vermont, Burlington.

HALL, P. (1977). The occurrence of disfluencies in language-disordered school-aged children. *Journal of Speech and Hearing Disorders, 42,* 364–86.

HUTT, D. (1985). *Mother-child differences in rate of speech: A pilot study.* M.A. Thesis, Temple University.

HUTT, D. (1987). *Mother-child differences in rate of speech in Spanish-speaking families.* Poster session, ASHA.

INGHAM, R. (1984). *Stuttering and behavior therapy: Current status and experimental foundations.* San Diego, CA: College-Hill Press.

JAYARAM, M. (1984). Distribution of stuttering in sentences: relationships to sentence length and clause position. *Journal of Speech and Hearing Research, 27,* 338–41.

JOHNSON, W. (1944). The indians have no word for it. Stuttering

in children. *Quarterly Journal of Speech, 30,* 330–37.

JOHNSON, W. (1955). A study of the onset and development of stuttering. In W. Johnson & R. R. Leutenegger (Eds.), *Stuttering in children and adults.* Minneapolis: University of Minnesota Press.

JOHNSON, W. (1961). *The onset of stuttering.* Minneapolis: University of Minnesota Press.

KENT, R., & FORNER, L. (1980). Speech segment duration in sentence recitations by children and adults. *Journal of Phonetics, 8,* 157–68.

KLATT, D. (1975). Vowel lengthening is syntactically determined in connected disclosures. *Journal of Phonetics, 3,* 129–40.

KLINE, M. L., & STARKWEATHER, C. W. (1979). *Receptive and expressive language performance in young stutterers.* Convention address, ASHA.

KEUHN, D., & TOMBLIN, J. B. (1974). *The use of cineradiographic techniques for the study of articulation disorders.* Convention address, ASHA.

KIDD, K. (1980). Genetic models of stuttering. *Journal of Fluency Disorders, 5,* 187–201.

KRASHEN, S. (1973). Lateralization, language learning, and the critical period; some new evidence. *Language Learning, 23,* 63–74.

MALLARD, R. (1988). *Family intervention: A British-American cooperative effort.* The Second Oxford Dysfluency Conference, Oxford, England.

MERITS-PATTERSON, R., & REED, C. (1981). Disfluencies in the speech of language-disordered children. *Journal of Speech and Hearing Research, 46,* 55–58.

MEYERS, S. (1988). *The University of Virginia Fluency Development Clinic: Diagnosis-Treatment-Research with preschool stutterers and parents.* The Second Oxford Dysfluency Conference, Oxford, England.

MEYERS, S., & FREEMAN, F. (1985a). Interruptions as a variable in stuttering and disfluency. *Journal of Speech and Hearing Research, 28,* 428–35.

MEYERS, S., & FREEMAN, F. (1985b). Mother and child speech rates as a variable in stuttering and disfluency. *Journal of Speech and Hearing Research, 28,* 436–44.

PETERS, H. F. M., HULSTIJN, W., & STARKWEATHER, C. W. (1989). Acoustic and physiological reaction times of stutterers and nonstutterers. *Journal of Speech and Hearing Research, 32,* 668–680.

PINDZOLA, R. (undated). *Stuttering intervention program: Age 3 through Grade 3.* Unpublished manuscript, Auburn University.

PRINS, D. (1984). Treatment of adults: Managing stuttering. In R. Curlee & W. Perkins *Nature and treatment of stuttering: New directions.* San Diego, CA: College-Hill Press.

RILEY, G., & RILEY, J. (1979). A component model for diagnosing and treating children who stutter. *Journal of Fluency Disorders, 4,* 279–93.

RUSTIN, L. (1987). The treatment of childhood dysfluency through active parental involvement. In L. Rustin, H. Purser, & Rowley, D. (Eds.), *Progress in treatment of fluency disorders.* London: Taylor & Francis.

RUSTIN, L., PURSER, H., & ROWLEY, D. (Eds.). (1987). *Progress in the treatment of fluency disorders.* London: Taylor & Francis.

RUSTIN, L., & PURSER, H. (1988). *An investigation of childhood and adolescent stutterers.* The Second Oxford Dysfluency Conference, Oxford, England.

SHEEHAN, J. G. (1974). Stuttering behavior: A phonetic analysis. *Journal of Communication Disorders, 7,* 193–212.

SHINE, R. (1984). Assessment and fluency training with the young stutterer. In M. Peins (Ed.), *Contemporary approaches in stuttering therapy.* Boston: Little, Brown.

SLIS, I., HASELAGER, G., & RIETVELD, A. C. M. (1988). *Development of speech rate in Dutch children.* Unpublished manuscript, Institute of Phonetics, University of Nijmegen.

SODERBERG, G. (1966). The relations of stuttering to word length and word frequency. *Journal of Speech and Hearing Research, 9,* 584–89.

ST. LOUIS, K., & WESTBROOK, J. (1987). The effectiveness of treatment for stuttering. In L. Rustin, H. Purser, & E. Rowley, (Eds.). *Progress in the treatment of fluency disorders.* London: Taylor & Francis.

STARKWEATHER, C. W. (1981). Speech fluency and its development in normal children. In N. Lass (Ed.), *Speech and language: Advances in basic research and practice,* Vol. 4. New York: Academic Press.

STARKWEATHER, C. W. (1987a). *Fluency and stuttering.* Englewood Cliffs, NJ: Prentice-Hall.

STARKWEATHER, C. W. (1987b). The development of fluency in children. In *Proceedings of the 1983 Conference on Motoric and Linguistic Aspects of Stuttering,* Northwestern University, Evanston, IL. Memphis, TN: Speech Foundation of America.

STARKWEATHER, C. W. (1989). Motoric variations in the speech of nonstutterers at locations predictive of stuttering. In R. Schreuder (Ed.), *Interfacultaire Werkgroep van Taal- en Spraak-gedrag.* Max Planck Institute, University of Nijmegen, The Netherlands.

STARKWEATHER, C. W., & GOTTWALD, S. R. (1986a) Stuttering. In L. Cole (Ed.), *The ASHA manual on the prevention of speech and language disorders.* Rockville, MD: American Speech-Language-Hearing Association.

STARKWEATHER, C. W., & GOTTWALD, S. R. (1986b). *Predicting the duration of treatment from information taken at intake.* Convention address, ASHA.

STARKWEATHER, C. W., GOTTWALD, S. R., & HALFOND, M. M. (1990). *Stuttering prevention: A clinical method.* Englewood Cliffs, NJ: Prentice-Hall.

STEPHENSON-OPSAL, D., & BERNSTEIN-RATNER, N. (1988). Maternal speech rate modification and childhood stuttering. *Journal of Fluency Disorders, 13* (1) 49–56.

TASSIELLO, M. (1975). *Duration of /s/ in five and seven year olds.* M.A. Thesis, Hunter College.

UMEDA, K. (1975). Vowel duration in American English. *Journal of the Acoustical Society of America, 58,* 434–45.

UMEDA, K. (1977). Consonant duration in American English. *Journal of the Acoustical Society of America, 61,* 846–58.

VAN RIPER, C. (1973). *The treatment of stuttering.* Englewood Cliffs, NJ: Prentice-Hall.

WALL, M. J. (1977). *The location of stuttering in the spontaneous speech of young child stutterers.* Doctoral dissertation, CUNY.

WALL, M. J., & MYERS, F. L. (1984). *Clinical management of childhood stuttering.* Baltimore: University Park Press.

WALL, M. J., STARKWEATHER, C. W., & CAIRNS, H. (1981). Syntactic influences on stuttering in young child stutterers. *Journal of Fluency Disorders, 6,* 283–98.

WATSON, B., & ALFONSO, P. (1987). Physiological bases of acoustic LRT in nonstutterers, mild stutterers, and severe stutterers. *Journal of Speech and Hearing Research, 30,* 434–47.

WEBSTER, R. L. (1974). In K. Calhoun, H. Adams, & K. Mitchell (Eds.), *Innovative treatment methods in psychopathology.* New York: Wiley.

Chapter 11

RESEARCH ON STUTTERING TREATMENT FOR ADULTS AND ADOLESCENTS: A PERSPECTIVE ON HOW TO OVERCOME A MALAISE

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The general purpose of this paper is to highlight some areas of research that appear to be fertile and necessary if stuttering treatment for adults and adolescents is to advance towards a reliable and credible clinical practice. These observations largely stem from recent reviews of stuttering treatment reports (Bloodstein, 1987; Curlee & Perkins, 1984; Hegde, 1985; Ingham, 1984; Ingham, in press; Peins, 1984) that have also directed attention to some therapy procedures and issues that appear to merit continuing investigation. These recent reviews provide a satisfactory account of the "state-of-the-art," and so it would serve little purpose to review the reviews. Instead, this paper focuses on a hierarchy of issues that have emerged from that literature.

Two general observations about stuttering treatment for adults and adolescents can be gleaned from the reviews of stuttering therapy that colleagues and this writer have conducted (Ingham, 1984; Ingham, in press; Ingham & Andrews, 1973; Ingham & Lewis, 1978). Firstly, the major developments have been spawned by behavior therapy procedures which underlie most of the practices that clinicians are now trained to use (Curlee, 1986). Secondly, the research activated by those practices has shown a sharp decrease over the past five years (Ingham, in press).[1] There appear to be three broad reasons for this situation: (a) the uncertain validity of therapy outcome evaluation procedures; (b) the unspecified and even undefined constituents of most therapy programs; and (c) the enormous cost of carefully controlled therapy evaluation studies. The first two of these reasons, which essentially refer to treatment outcome and treatment process issues, will form the foundation of this paper. If these two reasons could be resolved then the third should be a primary concern of funding agencies such as the National Institutes of Health.

Treatment Outcome Issues

The starting point for the current malaise in research on stuttering therapy must surely be the lack of agreement, and lack of research, on what is necessary in an adequate evaluation of stuttering therapy. There is probably a modicum of agreement that for treatment evaluation research to have any form of internal and external validity, it should be based upon repeated sampling of speech behavior across speaking situations and at intervals before, during and after treatment. The agreed measures of that speech appear to be frequency of stuttering events and speaking rate; but beyond that, there is virtually no consensus. There have been some efforts made to formalize the constituents of that format (Bloodstein, 1987; Ingham, 1984; Ingham & Costello, 1985), but most of these have not been derived from systematic research. Even more concerning, however, is the virtual absence of evidence showing the relationship between variations in stuttering and speech rate measures and their clinical relevance. For instance, there is no agreement on the combination of changes in these measures that are relevant to therapeutic improvement or, presumably, the target of normally fluent speech. In turn, of course, this has made it increasingly necessary to find the principal and functional constituents of normally fluent speech. That search might also locate variables that should be modified if stuttering therapy is directed towards the achievement of normally fluent speech (Finn & Ingham, 1989). However, the difficulties do not end there! It is far from clear that just because stutterers might achieve stutter-free and even perceptually normal speech that their speech is also acoustically, physiologically, and experientially normal. And even data derived from those dimensions would not begin to address the issue of durability.

[1]Actually, over the past five years, there has been quite a dramatic decline in the number of data-based investigations or reports on or concerning stuttering therapy for adults or adolescents. A general review of all English language journal publications that fit this category shows that in 1973–77 there were 57 reports, in 1978–82 there were 50 reports, but in 1983–87 (and including half of 1988) there were only 25 reports.

What, therefore, do clinical researchers need in order to assess the effects of stuttering therapy? One obvious answer, it seems, is for research to provide an assessment technology which is built on a foundation of reliable and valid measures of dimensions that identify stuttering and will reflect the changes that should occur in order to demonstrate therapy benefits. That technology should be based on a clear understanding of the constituents of normal speech production (at the physiological, acoustical, and perceptual level) if it is ever going to be possible to provide clinicians with a viable assessment technology. Conceivably, a physiological and acoustical analysis system might be developed so as to determine whether the speaker has speech production skills that fall within a target domain of normalcy. The system might then specify the dimensions that require modification to achieve that target domain. This is predicated on the assumption that the ultimate therapy objective is normally fluent speech including the absence of concern about its fluency) and that this is only possible if "normal" speech production is possible. This assessment technology would obviously emerge from current investigations into the speech motor system of stutterers and goes well beyond some existing procedures that appear to suggest that variations in stuttering frequency correspond to variations in the disorder. It does, however, incorporate a critical role for perceptual analyses (made by the speaker and observer) which, as Onslow and this writer recently argued (Onslow & Ingham, 1987), must accompany any acoustic and/or physiologic depiction of fluency.

Investigations designed to develop an adequate outcome evaluation technology should also attempt to identify the necessary requirements for valid and reliable speech sampling. There is a dire need for empirical studies that might establish guidelines for determining the frequency, duration and circumstances in which stutterers' speech should be sampled in order to depict the disorder and to reflect therapy benefits. If procedures could be developed to identify "pivotal" speaking situations (that is, situations which act as "barometers" for all or most speech performance), then this might not only reduce the magnitude or outcome evaluation, but also provide useful settings for therapy—or, better yet, settings that might be "manufactured" in clinic settings.

The measurement of stuttering has been considered in another paper in this conference, so comments on this topic will be brief and only relevant to treatment evaluation. One succinct observation does seem to be warranted: it is almost scandalous that there are still no standard procedures for measuring stuttering reliably. The "moment of stuttering" continues to be as ill-defined and as unreliably measured as it has been ever since Johnson and Knott (1936) first promoted its use in our field (see Young, 1984). Clinical researchers have only contributed more problems (especially for replicability) by continuing to use "disfluencies," "dysfluencies" or "stutterings" as synonymous dependent variables (Wingate, 1984). Furthermore, there have been no systematic investigations into the methods that might improve the reliability with which these events can be counted. The issue is made even more concerning with Kully and Boberg's (1988) finding that different clinics markedly disagree in counting these events. It is of interest, therefore, that most areas of behavioral research have also found event counting or recording to be less reliable and viable than interval judgments (Baer, Wolf, & Risley, 1987). That would suggest a need for research that might determine whether judgments of different time intervals of speech as stutter-free/fluent/normally fluent, etc., can be made with more reliability than stuttering counts, and have equivalent, or, even better, clinical validity. It may well be, for instance, that such a measurement system will blend easily with speech naturalness ratings (Martin, Haroldson, & Triden, 1984), a measure that can be made reliably on brief intervals of speech and also has demonstrable clinical value (Ingham, 1985; Onslow & Ingham, 1987). Of course, this perceptual rating system for evaluating and modifying speech quality may also form a valuable link between clinically viable measures and the critical physiologic/acoustic dimensions that are relevant to normally fluent speech.

The recent evidence that speech naturalness ratings (Martin, Haroldson, & Triden, 1984) may be made with high levels of reliability by clinicians (Ingham, Martin, Haroldson, Onslow, & Lency, 1985) and, more intriguingly, by stutterers while speaking (Ingham, Ingham, Onslow, & Finn, 1989) offers considerable promise for treatment process and outcome research. This is especially so with respect to the search for a measure of levels of self-monitoring of speaking, or attention to speaking, by treated stutterers. This is a dimension of speech behavior that some consider important to the concept of normally fluent speech, and thereby relevant in evaluating the outcome of therapy (Bloodstein, 1987). Needless to say, this is an area worthy of further research.

One of the major concerns in treating stuttering in adults or adolescents appears to be the extent to which current therapy practices can be expected to produce an "absolutely normal speaker." In other words, is total recovery possible using nonphysiological or behavioral treatments for what many believe to be a genetically determined neurophysiological disorder? This concern also appears to hover behind recent claims that some stutterers should be considered incurable (Cooper, 1987), and that behavior change has inconsequential therapeutic implications. It is likely that this issue has also emerged because of the paucity of research on current therapy procedures (a point that will be considered later in this paper), but it may also stem from the lack of knowledge about the entire process of recovery, especially the rate and extent of change that is possible in the speech behavior of adults or adolescents who have stuttered since childhood. For this reason, there may be much to be gained from carefully organized investigations of persons who have recovered from stuttering, either during their childhood, adolescence, or adulthood.

Numerous surveys (see Wingate, 1976) have established that there are large numbers of adults who claim to have used their own resources to recover completely from stuttering. These recoveries are reported to occur at

different ages and after varying periods with the disorder. The systematic investigation of the speech behavior of these persons would appear to be extremely useful for many reasons. It would establish whether their recovery has indeed resulted in speech that is indistinguishable from normal fluency (physiologically, acoustically, perceptually, and experientially), and if it is not, then such research might identify variables that form the residue of stuttering or, worse yet, a chronic foundation that is unlikely to change despite years of using stutter-free speech; it would also help to delineate the limits to recovery that result from years of stuttering or nonphysiological intervention. Of course, such investigations may also help to identify treatment variables that may help stutterers achieve durable stutter-free speech. From the perspective of the current zeitgeist regarding the foundations of stuttering, it would seem that the characteristics of recovered stutterers' speech motor systems (provided their speech is perceived and experienced as normal) might yield answer to questions concerning the structural or functional chronicity of stuttering. Of course, these findings would have profound implications for stuttering therapy. At the very least, they might show whether complete recovery is indeed a reality. It almost goes without saying that recovered stutterers might form one of the more relevant control groups in evaluating therapy outcome.

Treatment Method Research Directions

It is difficult to know where to begin in trying to identify important areas of research on therapy for adolescent or adult stutterers. Unfortunately, many areas require resolutions to the measurement and evaluation issues mentioned in the previous section, and many depend on clearer specifications about the purpose of stuttering treatment (Ingham, 1985). Nevertheless, the following is an attempt at highlighting some areas that appear to be in desperate need of research if there is to be any improvement in the quality, efficacy, and reliability of stuttering treatment for adults and adolescents. The areas appear to fall into two categories: (a) research on procedures that are designed to establish normally fluent speech, and (b) research on procedures that are designed to generalize and maintain therapy gains.

There can be little doubt that since the early 1970s, the most favored treatments for stuttering in adults and adolescents have been based on training stutterers to use prolonged speech, or one of the burgeoning variants of this speech pattern, in order to modify stuttering (Bloodstein, 1987; Hegde, 1985; Ingham, 1984; Ingham, in press). Invariably, this procedure is used in a context utilizing contingency management principles, thereby marrying two well-established methods for reducing stuttering behavior (Ingham, 1984). These procedures are utilized in various formats, but increasingly their use is favored in an intensive setting. If usage (see Bloodstein, 1987; Ingham, 1984) and some very questionable conclusions from a meta-analysis investigation of stuttering therapy (Andrews, Guitar, & Howie, 1980) are any guide, then there is little doubt that these are the most clinically useful procedures. At the same time, however, it should embarrass a profession that prides itself on accountability and its advocacy of science to know that there is virtually no reliable information on the definitive or functional constituents of prolonged speech. This speech is characterized by vague descriptions, such as extended voicing, soft contacts, gentle onsets, reduced speech rate, and some acoustically and/or physiologically based systems (Agnello, 1975; 1987; Webster, 1977) for controlling behaviors that are presumed to fit these descriptors. However, with the exception of some very preliminary studies (Agnello, 1987; Ingham, Montgomery, & Ulliana, 1983), no serious attempt has been made to identify the necessary parameters in this speech pattern or to determine their role or potential in producing normal sounding stutter-free speech. Of course, this situation has also meant that it is impossible to make valid comparisons among the therapy programs, let alone replicate their procedures.[2] Indeed, it is this situation that appears to have led to the decline in stuttering therapy research, the emergence of dubious practitioners (see Ingham, 1984, Chapter 12), and recurring controversies over the effectiveness of therapy (Gregory, 1979; Ryan, 1985).

There are justifiable reasons for arguing that a research program directed towards the identification of the functional variables among speech patterns that modify stuttering could still provide the most profitable approach to the treatment and understanding of this disorder. There is a widespread and often overstated claim that therapy research on communication disorders should always be couched within or driven by a theoretical framework. If this principle was applied to the relief of pain, then the discovery of the beneficial, though theoretically detached, effects of acetylsalicyclic acid (aspirin), might have had little interest to clinical researchers. Instead, a pragmatically driven interest in the effectiveness of this compound has led, first, to a reliable agent of treatment and, only later, to theoretically acceptable explanations for its action (cf. Lasagna & McMahon, 1983). Thus, in spite of the uncertainty about the parameters or variables that constitute prolonged speech (Hegde, 1985; Ingham, 1984), its almost undisputed effectiveness in reducing stuttering (especially in comparison with other procedures) makes it an area that almost cries out for research.

[2]A perfect example of this situation is described in a recent study by Waterloo and Gotestam (1988, p. 14) evaluating the efficacy of Azrin and Nunn's (1974) "regulated breathing method" for treating stuttering in adults. The vague descriptions of this method indicate that it relies on a variant of prolonged speech. Waterloo and Gotestam report that the treatment procedures in their study were based on consultations that occurred between S. Olaussen and Azrin in order to obtain a description of the method. This provided an unpublished description that Waterloo and Gotestam had to use to "replicate" Azrin's method, a method not described in replicable form in any publication. Needless to say, Waterloo and Gotestam's study also cannot be replicated.

At the very least, such research might immediately and drastically improve the quality of stuttering therapy.

There are other therapy procedures that deserve additional consideration as profitable areas of research. For instance, the parameters and effects of contingency management procedures, especially time out, are still poorly understood (see Prins & Hubbard, 1988) and have yet to be systematically investigated for their relationship to speech pattern procedures. Perhaps similar arguments could be advanced for chorus reading, although there is little doubt that contingency arrangements have much more clinical viability, particularly when they are self-managed. The principles that are used to control contingency management program may still underlie most of the functional variables that occur in stuttering therapy.

The second critical area of need for therapy process research relates to procedures that are designed to generalize or maintain therapy gains. Much of this area of research will also require solutions to the issues mentioned earlier in this paper. It should be obvious that until there is some agreement about the variables in the therapy process that must change in order to procure normally fluent speech, then there will continue to be serious impediments to generalization research. Equally handicapping, of course, are the enormous time demands and sampling condition requirements that are necessary to provide clinically meaningful findings in generalization/maintenance research (see Ingham, 1984; 1985). Nonetheless, there are some potentially profitable areas that researchers should pursue if this major therapy problem is to be surmounted.

At least three lines of research seem to have potential in order to increase the durability of treatment gains resulting from appropriately specified and demonstrably effective procedures. The first is through careful research on performance-contingent schedules that are designed to reinforce maintenance. At least two studies (Ingham, 1980; 1981) with adult stutterers treated in intensive prolonged speech programs have shown that such schedules can increase the probability that therapy gains are sustained and that they do generalize. A second profitable line of research would incorporate investigations of self-management schedules, such as those that have been demonstrated to be effective with response-contingent time-out (James, 1982; Martin & Haroldson, 1982) or prolonged speech (Ingham, 1982). A third potentially profitable line of research should emerge from the growing cognitive-behavioral perspective on generalization and maintenance which is largely based on self-efficacy theory (Bandura, 1977). From this pespective, Kirschenbaum and Tomarken (1982) have derived principles for effective treatment generalization that apply to many disorders. Such principles may be extremely relevant to stuttering therapy.

In addition to these essentially behavioral-cognitive approaches to stuttering therapy, there are mounting reasons for closely examining the effects of various drugs that have been reported to ameliorate stuttering. These include bethanecol chloride (Hays, 1987), carbamazepine (Goldstein, 1987), propranolol (Cocores, Dackis, Davies, & Gold, 1986), and imipramine (Boberg, personal communication). All of these reports have been accompanied by little more than the most rudimentary therapy evaluation. Claims for the efficacy of these drugs deserve careful investigations in order to prevent a repetition of the sorry saga of haloperidol's "contribution" to stuttering treatment (Ingham, 1984).

Finally, there is a growing amount of interest in the assessment and treatment of adult onset of stuttering (see Helm-Estabrooks, 1986). There are some intriguing and controversial reports about the relevance of some adult onset cases to current neurological perspectives on the disorder (Helm-Estabrooks, Yeo, Geschwind, Freedman, & Weinstein, 1986), but such accounts are also poorly documented (Ingham, 1987). That is also true with respect to therapy for adult onset stuttering (Ingham, in press). There is an undoubted need for research that will identify the factors that do and do not distinguish between an acquired and a developmental stuttering. The problems in this area are obviously much related to the evaluation issues mentioned in the early part of this paper. In sum, there seems to be a number of prominent research areas that merit consideration if treatments for adult and adolescent stutterers are to be advanced. To some extent, these areas can be ranked because some presuppose solutions in other problematic areas. At the origin of this ranking is the need for the development of methods and measures that are suitable for treatment outcome evaluation. This is especially true with respect to the dimensions of normally fluent speech. There may also be much that can be gained from a careful investigation of recovered stutterers. The resolution to some of these issues might make it possible to satisfy an almost overwhelming need for investigations into the therapeutic constituents of prolonged speech or variants of this speech pattern. Of almost equal concern is the need for generalization/maintenance research. There are, of course, other areas that deserve attention; some have been mentioned, while others have been mentioned in various reviews on stuttering therapy (see Dalton, 1983; Hegde, 1985; Shames & Rubin, 1986). However, the widespread use and ill-understood components of prolonged speech treatments appear to make this the most immediately important area for research in order to overcome the malaise that currently surrounds stuttering treatment research.

REFERENCES

AGNELLO, J. G. (1975). Voice onset and voice termination features of stutterers. In L. M. Webster (Ed.), *Proceedings of the Hayes Martin Conference on Vocal Tract Dynamics in Stuttering*. New York: Speech and Hearing Institute.

AGNELLO, J. G. (1987). A comprehensive computer program for facilitating fluency in stutterers. In H. F. M. Peters & W. Hulstijn (Eds.) *Speech motor dynamics in stuttering* (pp. 307–315). New York: Springer-Verlag.

ANDREWS, G., GUITAR, B., & HOWIE, P. (1980). Meta-analysis of the effects of stuttering treatment. *Journal of Speech and Hearing Disorders, 45*, 287–307.

AZRIN, N. H., & NUNN, R. G. (1974). A rapid method of eliminat-

ing stuttering by a regulated breathing approach. *Behaviour Research and Therapy, 12,* 279–286.

BAER, D. M., WOLF, M. M., & RISLEY, T. R. (1987). Some still-current dimensions of applied behavior analysis. *Journal of Applied Behavior Analysis, 20,* 313–327.

BANDURA, A. (1977). Self-efficacy: toward a unifying theory of behavioral change. *Psychological Review, 84,* 191–215.

BLOODSTEIN, O. (1987). *A handbook on stuttering.* Chicago: National Easter Seal Society.

BOBERG, E. (1988). Personal communication.

COCORES, J. A., DACKIS, C. A., DAVIES, R. K., & GOLD, M. S. (1986). Propranolol and stuttering. *American Journal of Psychiatry, 143,* 1071–1072.

COOPER, E. B. (1987). The Chronic Perseverative Stuttering Syndrome; incurable stuttering. *Journal of Fluency Disorders, 12,* 381–388.

CURLEE, R. F. (1985). Training students to work with stutterers. *Seminars in Speech and Language, 6,* 131–141.

CURLEE, R. F., & PERKINS, W. H. (EDS.). (1984). *Nature and treatment of stuttering: New directions.* San Diego: College-Hill Press.

DALTON, P. (ED.). (1983). *Approaches to the treatment of stuttering.* London: Croom Helm.

FINN, P., & INGHAM, R. J. (1989). The selection of "fluent" samples in research on stuttering: conceptual and methodological considerations. *Journal of Speech and Hearing Research, 32,* 401–418.

GOLDSTEIN, J. A. (1987). Carbamazepine treatment for stuttering. *Journal of Clinical Psychiatry, 48,* 39.

GREGORY, H. H. (ED.). (1979). *Controversies about stuttering therapy.* Baltimore: University Park Press.

HAYS, P. (1987 January). Bethanecol chloride in treatment of stuttering. *Lancet,* 271.

HELM-ESTABROOKS, N., YEO, R., GESCHWIND, N., FREEDMAN, M., & WEINSTEIN, C. (1986). Stuttering: Disappearance and reappearance with acquired brain lesions. *Neurology, 36,* 1109–1112.

HEGDE, M. N. (1985). Treatment of fluency disorders: state of the art. In J. M. Costello (Ed.), *Speech disorders in adults: Recent advances.* (pp. 155–188). San Diego: College-Hill.

INGHAM, R. J. (1980). Modification of maintenance and generalization in stuttering treatment. *Journal of Speech and Hearing Research, 23,* 732–745.

INGHAM, R. J. (1982). The effects of self-evaluation training on maintenance and generalization during stuttering treatment. *Journal of Speech and Hearing Disorders, 47,* 271–280.

INGHAM, R. J. (1984). *Stuttering and behavior therapy: Current status and experimental foundations.* San Diego: College-Hill Press.

INGHAM, R. J. (1985). Stuttering treatment outcome evaluation: closing the credibility gap. *Seminars in Speech and Language, 6,* 105–123.

INGHAM, R. J. (1987). Stuttering assessment and acquired brain lesions. *Neurology, 37,* 1434.

INGHAM, R. J. (in press). Stuttering. In A. S. Bellack, M. Hersen & A. E. Kazdin (Eds.), *International handbook of behavior modification and therapy.* New York: Plenum Press.

INGHAM, R. J., & ANDREWS, G. (1973). Behavior therapy and stuttering: A review. *Journal of Speech and Hearing Disorders, 38,* 405–441.

INGHAM, R. J., & COSTELLO, J. M. (1985). Stuttering treatment outcome evaluation. In J. M. Costello (Ed.) *Speech disorders in adults: Recent advances.* (pp. 189–223). San Diego: College-Hill Press.

INGHAM, R. J., INGHAM, J. M., ONSLOW, M., & FINN, P. (1989). Stutterers' self-ratings of speech naturalness: Assessing effects and reliability. *Journal of Speech and Hearing Research, 32,* 419–431.

INGHAM, R. J., & LEWIS, J. T. (1978) Behavior therapy and stuttering: and the story grows. *Human Communication, 3,* 125–152.

INGHAM, R. J., MARTIN, R. R., HAROLDSON, S. K., ONSLOW, M., & LENEY, M. (1985). Modification of listener-judged naturalness in the speech of stutterers. *Journal of Speech and Hearing Research, 28,* 495–504.

INGHAM, R. J., MONTGOMERY, J., & ULLIANA, L. (1983). The effect of manipulating phonation duration on stuttering. *Journal of Speech and Hearing Research, 26,* 579–587.

JAMES, J. E. (1981). Behavioral self-control of stuttering using time-out from speaking. *Journal of Applied Behavior Analysis, 14,* 25–37.

JOHNSON, W., & KNOTT, J. R. (1936). The moment of stuttering. *Journal of Genetic Psychology, 48,* 475–479.

KIRSCHENBAUM, D. S., & TOMARKEN, A. J. (1982). On facing the generalization problem: The study of self-regulatory failure. In P. C. Kendall (Ed.), *Advances in cognitive-behavioral research and therapy (Vol. 1)* (pp. 119–200). New York: Academic Press.

KULLY, D., & BOBERG, E. (1988). An investigation of interclinic agreement in the identification of fluent and stuttered syllables. *Journal of Fluency Disorders, 13,* 309–318.

LASAGNA, L., & MCMAHON, F. G. (EDS.). (1983). Proceeding of a symposium: New perspectives on Aspirin Therapy. *The American Journal of Medicine* (Special edition, June 14).

MARTIN, R. R., & HAROLDSON, S. K. (1982). Contingent self-stimulation for stuttering. *Journal of Speech and Hearing Disorders, 47,* 407–413.

MARTIN, R. R., HAROLDSON, S. K., & TRIDEN, K. A. (1984). Stuttering and speech naturalness. *Journal of Speech and Hearing Disorders, 49,* 53–58.

ONSLOW, M., & INGHAM, R. J. (1987). Speech quality measurement and the management of stuttering. *Journal of Speech and Hearing Disorders, 51,* 2–17.

PEINS, M. (ED.). (1984). *Contemporary approaches in stuttering therapy.* Boston: Little, Brown.

PRINS, D., & HUBBARD, C. (1988). Response contingent stimuli and stuttering: Issues and implications. *Journal of Speech and Hearing Research, 31,* 696–709.

RYAN, B. (1985). Training the professional. *Seminars in Speech and Language, 6,* 145–173.

SHAMES, G. H., & RUBIN, H. (EDS.). (1986). *Stuttering then and now.* Colombus: Charles E. Merrill.

WATERLOO, K. K., & GOTESTAM, K. G. (1988). The regulated-breathing method for stuttering: an experimental evaluation. *Journal of Behaviour Therapy and Experimental Psychiatry, 19,* 11–19.

WEBSTER, R. L. (1977). A few observations on the manipulation of speech response characteristics in stutterers. *Journal of Communication Disorders, 10,* 73–76.

WINGATE, M. E. (1976). *Stuttering: Theory and treatment.* New York: Irvington.

WINGATE, M. E. (1984). Fluency, disfluency, dysfluency, and stuttering. *Journal of Fluency Disorders, 9,* 163–168.

YOUNG, M. A. (1984). Identification of stuttering and stutterers. In R. F. Curlee & W. H. Perkins (Eds.), *Nature and treatment of stuttering: New directions* (pp. 13–30). San Diego: College-Hill Press.

G. SUMMARY

Chapter 12

RESEARCH DIRECTIONS IN STUTTERING: CONSENSUS AND CONFLICT

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Following presentation of the preceding manuscripts, workshop participants discussed the contents and related issues. Because these discussions expanded, clarified, and sometimes refuted what was presented, a summation of the discussions is warranted. On some of the issues, a consensus was reached; on others, unanimity was only approached; and on still others, opinion diverged. The essence and flavor of these discussions are presented below.

DEFINITION AND IDENTIFICATION OF STUTTERING

Some participants believed that a clear, precise definition of the disorder is needed, while others suggested that it is not, other than as a reliable diagnostic category. Potential components of a definition were offered, but consensus could not be reached. For research purposes, it is not clear how to decide precisely who is a stutterer. Factors presenting a consensus definition included:

- *Unknown etiology.* A consensus definition may require a better idea regarding what variable or variables cause the disorder of stuttering.
- *Levels of description and measurement.* The varying levels of description of stuttering (perceptual, acoustic, physiologic) contribute different and sometimes conflicting information (e.g., perceptually fluent speech may appear disordered using physiologic measures).
- *Group descriptions fail to consider individual differences.* It is unlikely that one measure could be identified (or developed) on which all stutterers would be impaired.
- *Variability.* Stuttered speech is highly variable over time and across different speaking situations. Such variability must be accounted for in a definition if it is to be useful for research purposes.

Defining stuttering may involve not only characterizing the number and nature of speech disfluencies, but also other speech behaviors, such as voice quality or naturalness. Some participants argued that whatever objective, listener-independent criteria are utilized to identify stuttering, subjective, listener-dependent elements will also play a role in any definition. Participants varied in their opinion regarding the value of self-identification (judgments of the adult stutterer) and/or parent-identifications (for the child stutterer).

One strongly suggested interim approach would be the explication of more precise, quantitative subject descriptors in research publications. Researchers might consider proposing a minimal set of parameters which should be used in describing subjects, parameters which might be utilized across studies. These might include frequency of disfluencies, type, duration, severity and clinical history of all subjects utilized in the study.

Information regarding normal fluency development is needed, as are investigations into differentiating normally dysfluent speech from mild stuttering, both in adults and children. Participants agreed that conducting such studies, in the absence of treatment, would be unlikely. To avoid an intervention bias, it was agreed that careful documentation or monitoring of treatment should be an essential component of such studies.

Participants discussed other directions which research might take in attempting to wrestle with the problem of definition. One proposal was simply to abandon such efforts. Some participants considered previous searches for a definition of stuttering as not useful and suggested that time would be better spent specifying the factors critical for the development and maintenance of stuttering, rather than in seeking a distillation of the "essence" of stuttering.

On the question of definition, still another idea was that investigating the behaviors of subjects who were at the extremes on the continuum, that is, speakers who are quite fluent, and speakers who are quite disfluent, would be one way to help clarify characteristics most closely related to stuttering as opposed to fluent speech. However, some participants were concerned that such an approach would fail to address or clarify the performance or behavior of subjects in the middle of the continuum. There was some agreement that studies are needed

which examine the value of "interval judgment", that is, deciding whether stuttering occurred within a given time frame (see "Measurement of Stuttering Severity"). Such investigations might address the ideal interval and how such a procedure would improve agreement concerning occurrence of stuttering. Work also is needed in determining the utility of such a procedure in studies of efficacy of treatment.

MEASUREMENTS OF STUTTERING SEVERITY

An objective measure of stuttering severity is needed. Such a measure should reflect the extent to which a person's ability to communicate effectively is impaired. However, participants could not reach consensus regarding *which* behaviors should be included in such a measure. Some participants agreed that sound/syllable repetition and sound prolongations should be included, but the role of other speech dysfluencies could not be agreed upon. Some participants supported the identification of *one* task, that would be representative of a variety of speaking situations. However, others were skeptical of this being feasible, claiming a variety of speech tasks would be necessary as no one task or situation could represent the speaking abilities of a stutterer. Such situations might include oral reading, alternating motion rate or inter-personal repetition, and speech communication. However, research is needed which addresses the meaningfulness of the differences between situations, as well as the reliability of the measurements used in the different situations.

Measurements of stutterers' speech was identified as a critical research need. One approach suggested was to utilize interval judgments, in which one sets the duration of time, and then determines the number and nature of stuttered syllables or words within that time period. Investigations are needed which evaluate such a procedure, determine how representative it is as a measurement of stuttering severity, and its validity and reliability. However, some participants acknowledged that use of this measure might result in a loss of specificity. For example, aspects of speech not specific to the problem of stuttering would be measured, thereby reducing valuable and specific diagnostic information.

Percent of speech which is disrupted was suggested as an alternative measure of severity, which might be supplemented with naturalness and speech quality ratings. Participants concurred that evaluation is also needed of the reliability and validity of *existing* methods of severity measurement. Regardless of measure utilized, participants concurred that several speech samples should be obtained, in view of variability in stuttering severity over time and across different speaking situations. The number and nature of samples which could be considered representative requires further study.

Measuring severity involves making judgments. In order to assure that those making the judgments are reliable, within and across situations, methods such as training to criterion should be utilized. Use of videotapes was recommended, in order to provide visual information which may be useful in the identification of stuttering.

ETIOLOGY

Identification of a single cause of stuttering is highly unlikely. Rather, a more appropriate direction is to identify phenomena which must be accounted for in a theory of stuttering. High density designs focused on families with one or more stutterers, and individuals with a history of stuttering, would be useful in identifying some of these phenomena. Relatives of stutterers were considered by the participants as an excellent pool for study, as a fertile area of research themselves, or to serve as a control group for studies of the affected members. In view of the heterogeneity of stuttering, some participants suggested that subtyping is an important precursor to addressing etiologic issues. Others felt it was more useful to focus on finding the multiple variables that underlie stuttering, while recognizing that these variables might be differentially weighted in each individual.

Participants concurred that developmental studies would have important implications to etiology. Parallel measurements of fluency, language, and other neurological or maturational factors would allow investigation of relationships between these areas, and address the possibility of a critical period of stuttering development.

Many of the participants believed that physiological measures of stuttered as well as fluent speech had been generally perceived as somehow "better" than other types of measures. It was acknowledged that measurements at many different levels of observation will be necessary to make significant progress in understanding the basis of stuttering.

SUBTYPING STUTTERERS

General enthusiasm was expressed for longitudinal studies of stuttering in children or children at risk for stuttering. Such an approach will be necessary before subtyping is possible, as there apparently are subtypes in the development of fluency. Participants considered a possible fruitful approach might be a longitudinal study of a group of at-risk and already diagnosed children. Factors could be identified which might differentiate those who would be likely to recover from those whose symptoms would persist. Factors to monitor might include manner and age of onset, frequency and nature of stuttering episodes, intellectual ability, language and articulation skills, and other behavioral characteristics. Such an investigation would best be done across several sites.

Participants did not reach consensus regarding the value of genetic history for subtyping. Although stuttering seems to involve a genetic component, it is not clear yet just what is inherited, for example, a predisposition to

stutter, or a more general ability such as language skills, which might aid or hinder a child in dealing with stuttering. Participants noted the difficulty in obtaining a genetic profile that would satisfy the requirements of behavioral geneticists. There was agreement that identification of stuttering in family members should be based on direct, objective observation and not on second-hand subjective verbal reports by other family members.

Subtyping of adult stutterers must take into consideration therapy histories which may have played a role in determining the nature of the current speech patterns. Subtyping information may lie in addressing such issues as relapse and the speed with which the adult stutterer moves through the treatment program. Investigations involving simultaneous exploration of speech production by stutterers and fluency perception by listeners are needed. Findings might elucidate the issue of whether fluency is a continuous or discontinuous function in production or in listener perception. Participants concurred that the integration of speech production studies with speech perception studies is particularly appealing and would be a productive approach into the study of stuttering.

A lack of understanding persists about the relationship of language disorders or language development to stuttering. This situation is complicated by less than complete knowledge regarding language development and speech motor control. Very little empirical research has been done on this connection. Participants agree that longitudinal studies would be important in addressing this issue.

It remains unclear why some children who begin to stutter in the preschool and early school years see their symptoms abate and disappear, while others continue to stutter for much longer periods of time. The issue of chronicity, and the critical period aspect of this question, deserve further investigation.

PATHOPHYSIOLOGY

The eventual understanding of the nature of stuttering may depend on a much greater understanding of the neural basis of speech motor control and hemispheric activation associated with linguistic and speech motor performance. Research into brain activation phenomena associated with speech movements under different conditions was suggested as a continuing research need. Participants recognized the need for a model which relates brain activities to speech production. However, participants agreed that improved spatio-temporal resolution in methods of studying human activity are needed before such modeling will be feasible. Careful specification of stimulus, tasks, and subject variables will be critical in such work.

Participants acknowledged that if stuttering research is to move forward, it will be important to attract scientists from a variety of relevant disciplines. The training of future researchers in stuttering must include improved and increased education in the neurosciences, along with training in the use of modern methods for analysis of the speech production process.

TREATMENT

Participants concurred that the present situation regarding treatment of stuttering is problematic. Therapy procedures are being promoted with little to no published data to support their effectiveness. In addition, there is little consistency across universities and clinics in the procedures utilized. There is a critical need for systematic evaluation of effectiveness and efficiency of early intervention (with at-risk or already stuttering children) and of treatment with adult stutterers. Clinical data are needed which support or refute the frequent claim that the age at entry into treatment is a critical factor in success (remission of symptoms).

The issue of relapse has obvious clinical significance, and implications for treatment. Studies of stutterers who have relapsed would be helpful and physiological data would assist in their examination. However, relapse may also address theoretical issues, for example, if we understand why stuttering recurs or returns, then we will have a better understanding of what stuttering is.

There is a need to characterize the treatment successes, or the "recovered" adult stutterers. Comparisons between these individuals and adults with no history of fluency problems will speak to such issues as persisting deficits (even in the face of "recovery") and the establishment of reasonable treatment goals. Participants concurred that there is a need for better specification of methods known to have a positive treatment effect with adult stutterers, such as "prolonged speech" or "gentle onset." Other potentially fruitful avenues of investigation with adult stutterers include drug studies and the role of self-management, and improved methods for self-collection of data.